ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

VOLUME 11
NUMBER 12
DECEMBER, 1958

OPERATIVE ORAL SURGERY

PRELIMINARY REPORT ON PLASTIC THROMBOPHLEBITIS IN THE PTERYGOCONDYLAR AREA IN THE EDENTULOUS PATIENT AND ITS CLINICAL AND PATHOLOGIC SIGNIFICANCE

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INTRODUCTION

SINCE Costen[1] first described his syndrome associated with malfunction of the temporomandibular articulation, various theories have been put forward to account for the symptoms associated with the joint dysfunction. In the main, the chorda tympani and auriculotemporal nerves have been implicated. Subsequent anatomists,[2] however, have demonstrated that the likelihood of the involvement of these nerves in joint displacement is improbable and that, furthermore, the symptoms are probably the consequence of a traumatic arthritis[3, 4] of the joint. However, the fact that many of our patients with local joint discomfort and reflex vascular disturbances, particularly of the upper extremities, have been relieved by the simple expedient of leaving their lower dentures out and have been permanently relieved by the restoration of the vertical dimension suggests that the symptoms might be caused by intermittent compression on some particular area of the joint and that the reflex vascular disturbances are caused by involvement of vessels in or near the joint, the reflex mechanism being similar to that described by one of us[5] on the basis of hyperactivity of sympathetic reflex arcs causing varying degrees of ischemia. In support of this contention, we have been able to demonstrate plastic thrombophlebitis in the pterygocondylar area. We believe that the plastic thrombophlebitis demonstrable in this strategically placed vessel is the result of intermittent compression during

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mastication. The elaboration of this thesis presents several facets. Therefore, we have thought it advisable to divide this article into three parts: clinical, anatomic, and pathologic.

CLINICAL CONSIDERATIONS

The patients were first seen at the outpatient department of the Royal North Shore Hospital in Sydney. They were examined and then referred to the neurological clinic and the dental clinic. At least 25 per cent of the edentulous patients attending the dental clinic of the Royal North Shore Hospital suffered reflex disturbances in some degree.

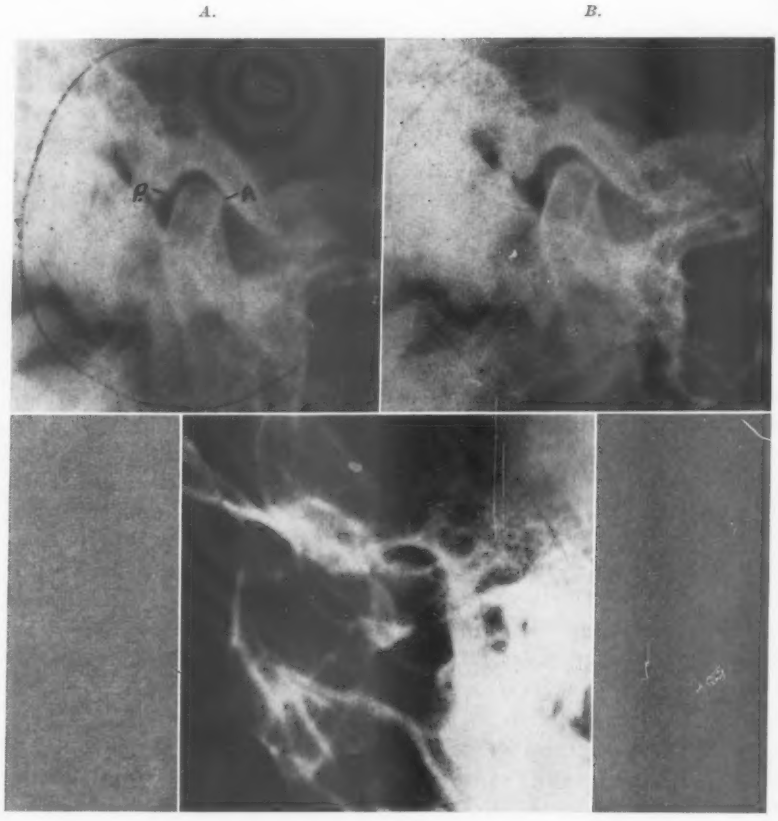


Fig. 1.—*A*, Pretreatment roentgenogram of the right temporomandibular joint of a female patient (her artificial teeth in habitual occlusion) demonstrating decrement of anterior disc space. The proportion of anterior disc space (*A*) to posterior disc space (*P*) is roughly 1:4.

B, Roentgenogram of the same patient without change of position, except that bite blocks have been inserted. There is increment of anterior disc space, and the proportions are roughly 1:2. The symptoms in this patient were numbness and pain, on the right side of the face, neck, and arms.

C, Roentgenogram of patient demonstrating gross decrement of posterior disc space. This patient had gross reflex vascular symptoms.

Symptomatology.—The patients generally presented with ipsilateral joint discomfort, although this was by no means the chief complaint. Pain in various parts of the head was a constant symptom. Varying degrees of hyperalgesia of the skin overlying the joint were always detectable. Paresthesia affecting various sites, in the main acroparesthesia, was always present. Vertigo, photophobia, tinnitus, and muscular hypertonicity were variable symptoms.

Diagnosis.—In all cases there was loss of vertical dimension, varying from slight to gross. The patients had been wearing the same dentures for ten to thirty years. When the patients were asked to occlude their teeth, the lower teeth were nearly always labial to the upper teeth. When questioned, the patients stated that when they first received their dentures the artificial teeth occluded in the normal overbite fashion. Roentgenograms of the temporomandibular joint with the artificial teeth in occlusion revealed decrement of disc space, either anteriorly, posteriorly, or superiorly. It was notable that anterior or posterior decrement of disc space could occur without superior decrement of disc space, but that the latter could not occur without decrement of either the anterior or the posterior disc space (Figs. 1 and 2).

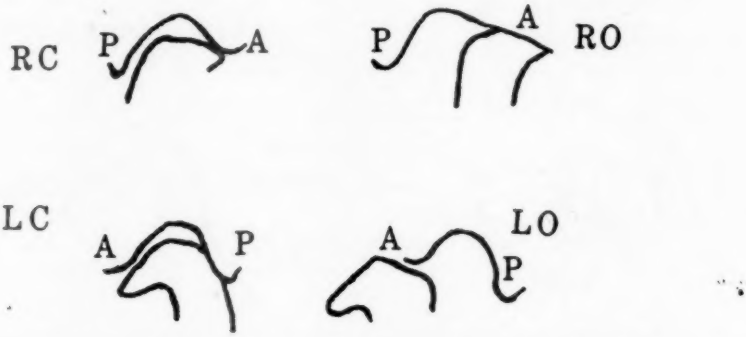


Fig. 2.—Roentgenogram tracings of the temporomandibular joint of a patient presenting gross loss of disc space and suffering from severe subjective symptoms, including acroparesthesia.

RC, Right temporomandibular joint with artificial teeth in occlusion.
RO, Right joint, mouth open.
LC, Left temporomandibular joint with artificial teeth in centric occlusion.
LO, Left joint, mouth open.
A, Anterior aspect of joint.
P, Posterior aspect of joint.

The patients experienced varying degrees of relief when instructed not to wear their lower dentures. The symptoms returned with their former severity when they reinserted their dentures. Two patients whose symptoms had hitherto been relieved by leaving out their lower dentures were instructed to leave them out except for the purpose of eating. When they did this, the symptoms continued unabated.

The assessment of disc space from roentgenograms is largely a matter of experience. We have used the Lindblom[6] technique to obtain a lateral projection

of the temporomandibular joint. There are several objections to this,[7] but nevertheless it fairly faithfully reproduces the anteroposterior position of the condyle in relation to the temporal bone. The articular fossa and the condyle vary from person to person and in the same person, but the radiolucent spaces between the condyle and the anterior and posterior surfaces of the articular fossa, with the teeth in occlusion, bear a fairly constant proportionate relationship to each other (Fig. 3).[8] It is probable that with loss of vertical dimension there is concomitant thinning of the articular disc in contact with the articular surfaces, and

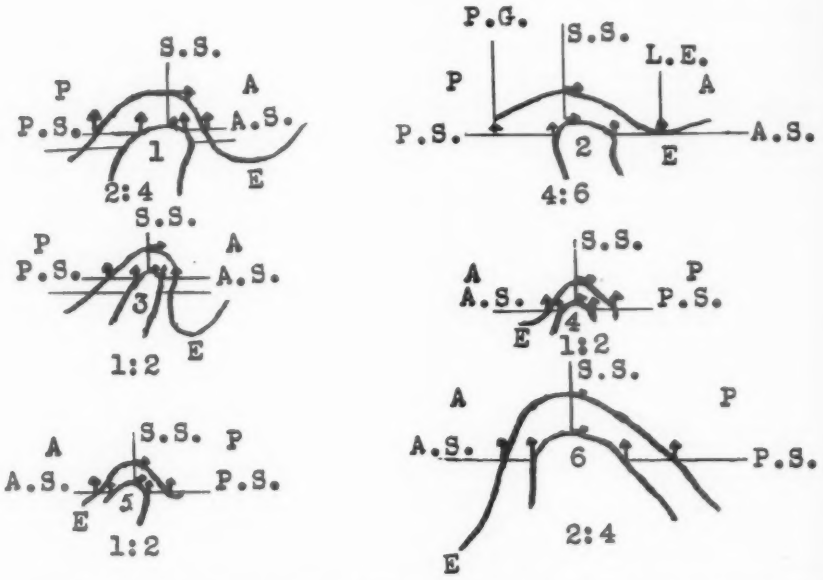


Fig. 3.—Tracings of roentgenograms and wax impression of normal temporomandibular joints with the teeth in centric occlusion. The lines between the arrows represent the respective disc spaces. The anterior disc space is assessed as the narrowest horizontal line between the anterior surface of the condyle and the anterior slope of the articular fossa (*A.S.*). The posterior disc space (*P.S.*) is assessed as the distance between the posterior surface of the condyle and the posterior surface of the articular fossa on a line horizontally continuous with the anterior line. This demonstrates the rough proportionate relationships between these respective distances.

1, Normal overbite. (After Benson.[9]) *2*, Edge-to-edge relationship. (After Benson.[9]) *3*, Deep overbite. (After Benson.[9]) *4*, Normal temporomandibular joint with teeth in centric occlusion. (After Salzmann: Principles of Orthodontics, ed. 2, Philadelphia, 1950, J. P. Lippincott Company.) *5*, Tracing of wax impression cut in sagittal plane with teeth in centric occlusion. (After Martin.[8]) *6*, Tracing of dissection. (After Chase.[12]) *A*, Anterior. *P*, Posterior. *A.S.*, Anterior disc space. *P.S.*, Posterior disc space. *S.S.*, Superior disc space. *E*, Articular eminence. *P.G.*, Postglenoid process.

this is seen on the roentgenograms as proportionate reduction of the radiolucent areas between the condyle and the temporal bone (Fig. 1). As this progresses, the articular surfaces of the condyle appear in the roentgenograms to be in contact with the articular fossa, both anteriorly and posteriorly (Fig. 2). The thinning of the disc may be uneven, however, so that in certain cases decrement of anterior or posterior disc space may be observed.

CASE 1.—The patient, Mrs. S., aged 67 years, was first seen at the outpatient department of the Royal North Shore Hospital on Aug. 4, 1954. She complained of pain in the neck, hip, and hands. Her hands and cervical and lumbosacral spine were x-rayed, and no abnormality was detected. Various therapeutic measures were instituted with no success. On Aug. 3, 1957, she again attended the outpatient department complaining of pain in both hands, the left more than the right, together with numbness and paresthesia. The pain became worse at night. On examination, no weakness was detected but there was apparent wasting of the left thenar eminence. There was mild hyperalgesia. Pressure on the left carpal tunnel produced pain. A provisional diagnosis of carpal tunnel syndrome was made. When she was seen at the dental clinic on Aug. 13, 1957, it was observed that there was gross loss of vertical dimension. It was also observed that the bite had been recorded in a protruded relationship. The temporomandibular joints were then x-rayed and the patient was instructed to leave her lower teeth out at all times. X-ray examination of her temporomandibular joints revealed that there was decrement of anterior disc space with her artificial teeth in habitual occlusion.

On Aug. 8, 1957, the patient was seen by the honorary neurologist, who reported: "Acroparesthesia of the left hand more than the right, at first nocturnal and now diurnal. She states emphatically that if she takes out her dentures she does not have the pain. She states that the middle finger on the left hand has felt dead on and off for fifteen years. She has had other symptoms for years, and she states that they are getting worse. Pain, fullness, and pins and needles are among the symptoms that she describes. She also thinks that all her fingers are involved but, when pressed, doubts if thumbs and little finger are involved. She has cervical spondylosis. On examination, there was no motor or sensory disturbance in the hand. Tendon reflexes are equal in each upper limb. She has been instructed to wear night splints and to observe the distribution carefully."

New dentures were constructed and the patient was educated in finding and using the correct centric occlusion during mastication. The position of her condyles was checked by x-ray examination with the teeth in centric occlusion.

On Sept. 18, 1957, the neurologist reported as follows: "She has been wearing the new dentures since her last visit and she has no further acroparesthesia. In fact, she says she has forgotten her hands. She also says that her head feels normal. In retrospect, she says that the fourth and fifth digits were sometimes involved but less than the others. There is no motor or sensory disturbance."

CASE 2.—The patient was Mrs. F., a 53-year-old woman. She was first seen at the outpatient department of the Royal North Shore Hospital on June 16, 1949, complaining

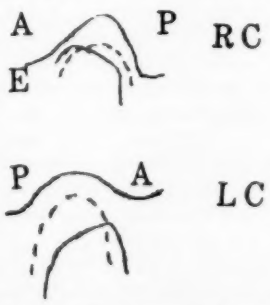


Fig. 4.—Superimposed roentgenograms of tracings of the patient in Case 2. The broken line represents the relationships of the condyles to the temporal bone, with her artificial teeth in centric occlusion after correction of the bite. The continuous line represents the position of her condyles before treatment. Note decrement of right disc space. In habitual occlusion prior to treatment the condylar relationships are consistent with right lateral excursion. It is probable that consistent habitual occlusion in this fashion accounts for the position of the right condyle. The condition could not be corrected without impinging further on posterior disc space, which already was less than one sees normally. However, we believe that posterior disc space is not as critical as anterior disc space.

of pain in the right hip. The radiologist reported that the appearance was consistent with Paget's disease of the right femur mainly of the amorphous variety, with some varus deformity, and that the lumbar spine showed no appreciable disease. At that time she also had psoriasis of the knees and elbows. When seen again on Sept. 10, 1957, the patient complained of pain in her right ear, which she had experienced on and off for four years. She complained of pain behind the eyes and on the right side of the face. She had acroparesthesia. She stated that, on leaving her dentures out, she did not experience any of the above symptoms. Roentgenographic examination of the temporomandibular joints revealed decrement of anterior disc space on the right side with her artificial teeth in occlusion, while on the left side, also with her artificial teeth in occlusion, the condyle was in an anteroinferior relationship to the articular fossa (Fig. 4). There was gross loss of vertical dimension. The new dentures were constructed, vertical dimension was restored, and the occlusion was balanced with subsequent disappearance of the symptoms (Fig. 4).

The following case is interesting from the symptomatic viewpoint:

CASE 3.—The patient was a 56-year-old woman, Mrs. R.

General history: She was first seen at the outpatient department of the Royal North Shore Hospital on Jan. 7, 1954, when she complained of a sore throat and soreness of the left ear. She was referred to the ear, nose, and throat clinic and was treated for acute tonsillitis. She was again seen on Sept. 17, 1956, when she still complained of soreness of the left ear. This time she also complained of a burning sensation of the mouth and tongue. On Oct. 22, 1956, the blood count was normal, but the patient further stated that she "had a funny feeling in the mouth, the nose, and even the eye." She also had "tingling in the foot." She has had shingles of the sciatic nerve. On June 12, 1957, the patient again presented and complained of pains at the back of the head, consistent with pulsation, and of intermittent nasal obstruction. On Aug. 2, 1957, she was examined again.

Previous symptoms: She had had these symptoms for the last three months preceding the August 2 examination. Previous to this, most of her sensations were confined to the mouth, for instance, a burning sensation that affected the roof of the mouth, lips, upper throat, right nostril, forehead, and right eye ("feels like a cold wind in the eye") and was intermittent but had improved slightly in recent months.

Present symptoms during last three months: These included "onset of pumping in right ear, pinging sound at the back of the head, bells in the right ear, a crackling sensation at the back of the neck for twelve months with walking and turning the head, and pain at the back and front of the head like a tight feeling." Taste and smell were unaffected. There were no deafness and no vertigo, but there was a sensation of a list to the right. She had had numbness in the arms for some years. Numbness in the left arm was greater than numbness in the right arm. The patient was never really free of "shunting of trains in the head." There was no undue dyspnea; she had a gastric ulcer twenty years ago; barium meal ten years ago was clear; the patient had had uterine prolapse. The patient seemed "nervous at times but not depressed." On examination, the patient was "obese, had a high color, the carotids were equal, discs were normal, neck movements full."

The provisional diagnosis was "presumably paresthesia of fifth nerve area can be correlated with other symptoms, but may all be functional." The patient was then directed to the neurology clinic for investigation. The honorary neurologist reported: "Right tinnitus for three months; clicking on moving jaws; wandering pains in head; burning in lips, tongue, and throat for one year; numbness of both arms and all of fingers at any time for years. Pain recently in left arm greater than right. On examination, very obese; blood pressure, 190/120; no abnormality in full neurological examination."

X-ray of skull and petrous bones: The pineal was calcified and central in position. Petrous bones appeared normal.

Neurologist's report (Aug. 8, 1957): "Burning in mouth, etc., is better in morning, as she does not wear dentures at night. Right tinnitus persists. Yawning makes tinnitus worse, as does exertion and moving head. On examination, cranial nerves intact. Blood pressure, 140/90."

The patient was first seen at the dental clinic on Sept. 19, 1957. There was gross loss of vertical dimension, and roentgenograms of the temporomandibular joints with the teeth in habitual occlusion revealed gross decrement of disc space.

Diagnosis: Temporomandibular joint syndrome with involvement of cephalic extensions of upper thoracic spinal nerves.

ANATOMIC CONSIDERATIONS

As previously stated, there is a considerable variation in the temporocondylar relationship of different persons and even in this relationship on opposite sides of the same person.[7] This may be determined, to a certain extent, by the bite. Benson[9] has represented the temporocondylar relationship in normal overbite, edge-to-edge bite, and deep overbite. Sagittal sections of the joint by Bauer,[10] Robinson,[11] and Chase[12] show similar variations (Figs. 5 and 6). From observations of roentgenograms of persons in the Lindblom position, using slight variation of the points suggested by Martin,[8] we have found a fairly constant proportional relationship (Fig. 3) between the anterior and posterior temporocondylar relationships. We consider the space between the anterior slope of the articular fossa and the anterior slope of the condylar head to be indicative of anterior disc space and the space between the posterior slope of the condylar head and the posterior slope of the articular fossa to be indicative of posterior disc space. We consider the anterior disc space to be rather critical, for clinical reasons already mentioned, and we also believe that the anatomic relationship of the hela of the pes menisci to the insertion of the lateral pterygoid muscle is of great significance.

We are further of the opinion that the superior orientation of the condylar head in the glenoid fossa in our patients is due to loss of vertical dimension, so that mastication occurs with the condylar head in an abnormal position in the glenoid fossa and, as a result of this, certain changes occur in the disc itself and in its relationship to the lateral pterygoid muscle.

Anatomy of the Disc.—The disc has been described by various authors. With the exception of Rees,[13] however, none have given particular names to particular parts. Rees has emphasized the varying thickness of the disc. He has divided the thickness of the disc into (1) an anterior moderately thick band, (2) a thin intermediate band, (3) a posterior thick band, and (4) a posterior bilaminar zone.

For the purpose of describing disc areas, however, we have thought it advisable to name the various parts, particularly as they appear on sagittal section. We have called the anterior moderately thick band the *pes,* the intermediate thin band the *pars gracilis,* the moderately thick posterior band the *pars posterior,* and the bilaminar zone the *parts bilaminar menisci* (Fig. 6).

The pes menisci: The pes menisci, as its name suggests, resembles a foot with the heel (hela) occupying the space between the insertion of the lateral

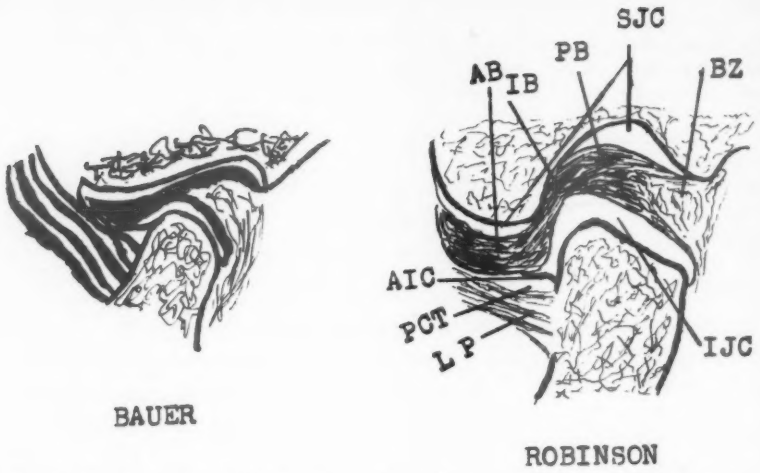
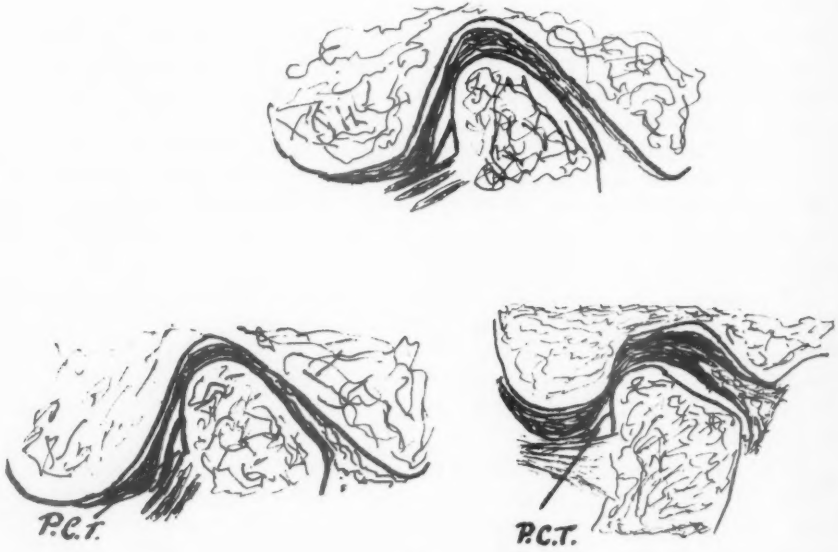


Fig. 5.—Tracings of sagittal dissections of the temporomandibular joint by Bauer and Robinson.

1, Sagittal dissection (by Bauer[10]). *2*, Sagittal dissection (by Robinson[11]). *AB*, Anterior moderately thick bands (Rees[13]). Pes menisci (authors). *IB*, Intermediate thin band (Rees[13]). Pars gracilis menisci (authors). *PB*, Posterior thick band (Rees[13]). Pars posterior menisci (authors). *BZ*, Bilaminar zone (Rees[13]). Pars bilaminar menisci (authors). *SJC*, Superior synovial cavity. *IJC*, Inferior synovial cavity. *AIC*, Anterior inferior portion of capsule. *PCT*, Pterygocondylar triangle. *LP*, Insertion of lateral pterygoid.

A.



B.

Fig. 6.—*A*, Tracing of a sagittal dissection by Chase.[12] This probably represents nearly the extreme limit of normalcy. The glenoid fossa is very concave, and the condylar head is located quite superiorly in the fossa.

B, Drawings show the difference in relationship of the condylar head in the dissection of the fossae by Bauer[10] and Chase[12] if, for pathologic reasons, the condylar head intruded more into the fossa and especially the compression of the pterygocondylar area (*P.C.T.*).

pterygoid muscle and the anterior slope of the articular head. The greater part of the inferior surface is in contact with the superior surface of the tendinous insertion of the lateral pterygoid muscle, while the tendons of that muscle that are attached to the disc pass into its inferior surface just anterior to the hela of the pes. The anterior extremity of the pes is attached to the anterior part of the articular tubercle, while its superior surface is separated from the fibrous covering of the articular slope by the superior synovial space. The relationship of the hela of the pes to the lateral pterygoid muscle and to the condyle deserves special consideration and, since it constitutes the superior medial surface of the pterygocondylar area, will be described in the discussion of that area.

Pars gracilis menisci: The pars gracilis menisci is the thin intermediate portion of the disc that joins the pes to the pars posterior. It is separated from the anterior slope of the articular head of the condyle by the inferior joint space and the fibrous covering of the condyle. Similarly, it is separated from the anterior slope of the articular fossa by the superior synovial space and the fibrous covering of that slope. It is relatively avascular and constitutes the anterior disc space (Fig. 7).

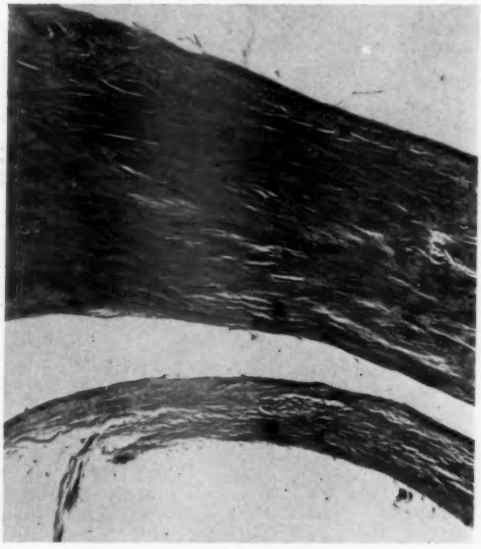


Fig. 7.—The pars gracilis menisci.

Pars posterior menisci: The pars posterior menisci is structurally similar to the pars gracilis and is separated in a similar fashion from the articular surfaces of the joint. It is the thickest part of the disc proper and is prolonged posteriorly to form the upper and lower strata of the pars bilaminar menisci. It is relatively avascular and constitutes the superior disc space.

Pars bilaminar menisci: The junction of the pars posterior and pars bilaminar menisci constitute the genu. The upper stratum of the pars bilaminar is prolonged posteriorly to be attached to the petro-tympanic fissure, the lower

Fig. 8.

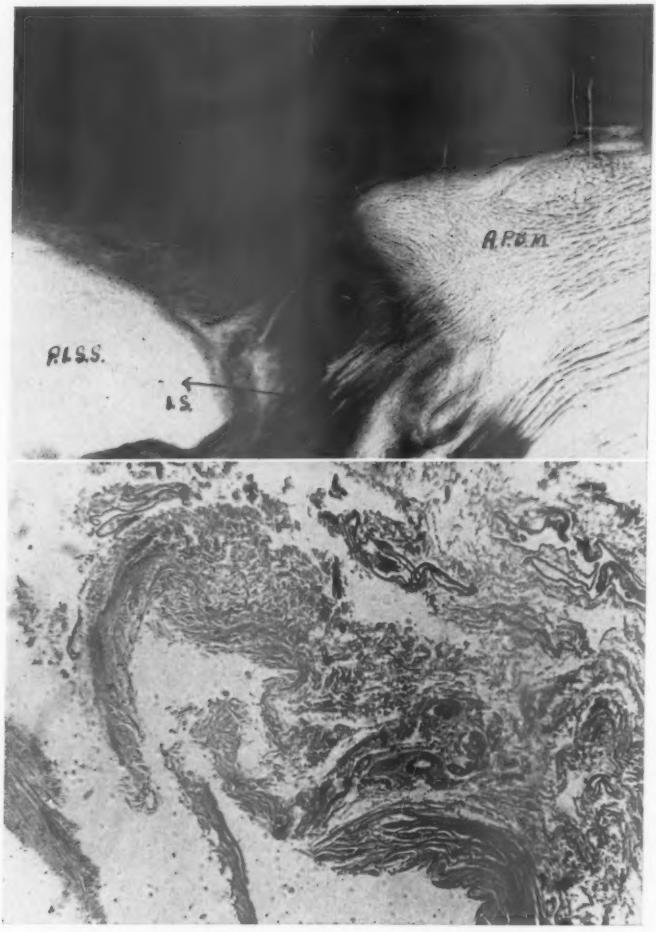


Fig. 9.

Fig. 8.—The pars bilaminar menisci.

P.I.S.S., Posterior inferior synovial space. *I.S.*, Inferior stratum. *A.P.B.M.*, Apex of pars bilaminar menisci.

Fig. 9.—Neurovascular bundle piercing the fibrous covering of the condyle just posterior to the insertion of the posterior inferior capsule of the joint.

stratum is prolonged posteriorly to be attached to the posterior slope of the condyle and the synovial spaces superiorly and inferiorly complete the synovial compartments. This area constitutes the posterior disc space (Fig. 8).

The sagittal crest of the condyle in the closed position is said to be just posterior to the genu. Between the upper and lower strata of the pars bilaminar the tissue becomes progressively less fibrous and more vascular, and numerous myelinated nerve fibers can be demonstrated. Just posterior to the attachment of the lower stratum to the fibrous covering of the condyle, a rather large neurovascular bundle can be seen piercing this structure and is probably an articular branch of the sympathetic system innervating the condyle (Fig. 9).

The pterygocondylar area: The pterygocondylar area is intimately related to the hela of the pes menisci. It is interposed between the attachment of the lateral pterygoid muscle to the neck of the condyle and the anterior inferior margin of the inferior synovial compartment of the joint (Fig. 6[2]). Its boundaries in sagittal section constitute a triangle. Its superior boundary in its medial half is the anterior inferior capsule and the hela of the pes menisci. In its lateral half its superior boundary consists of the superior fibers of the lateral pterygoid muscle. Its inferior boundary is composed of the inserting condylar fibers of the lateral pterygoid muscle, and its base, in its medial half, is between the origin of the anterior inferior part of the capsule and the tendon of the lateral pterygoid muscle. In its lateral half its base is the space between the insertion of a fine tendon of the lateral pterygoid muscle to the condyle and the main tendinous attachment. It is exceedingly vascular and contains several bundles of myelinated nerve fibers (Figs. 10 and 11).

When the teeth are occluded in edentulous patients with gross loss of vertical dimensions, in certain cases the condylar head is located superiorly anteriorly in the glenoid fossae with concomitant decrement of anterior disc space. When this occurs, closer approximation of the hela of the pes menisci to the pterygocondylar area is inevitable because of the superior reorientation of the insertion of the lateral pterygoid muscle. The pterygocondylar area is, in fact, compressed.

PATHOLOGIC CONSIDERATIONS

The specimen to be examined was dissected from a cadaver (aged 57, death due to carcinoma of the lungs) from which we had demonstrated radiographically a decrement of anterior disc space. The condylar head, together with the disc and the attachment of the lateral pterygoid, was removed from the cadaver.

Macroscopic.—The condylar head was normal in shape, the pars posterior menisci and the pes menisci also being relatively normal. The pars gracilis, however, appeared exceptionally thin. The disc was cut in serial sagittal sections at 10-micron intervals. A vein located in the pterygocondylar area exhibited excessive thickening of one of its walls. We followed this in serial section to be sure that the thickened appearance was not due to the plane of cutting. We concluded that this was not the case since, if it were, at some stage of its length commensurate thickening of the opposite wall should be seen. The thickened part of the vein consisted of fibrous tissue, and the cells did not exhibit any signs of degeneration. It was therefore considered that the vein exhibited plastic

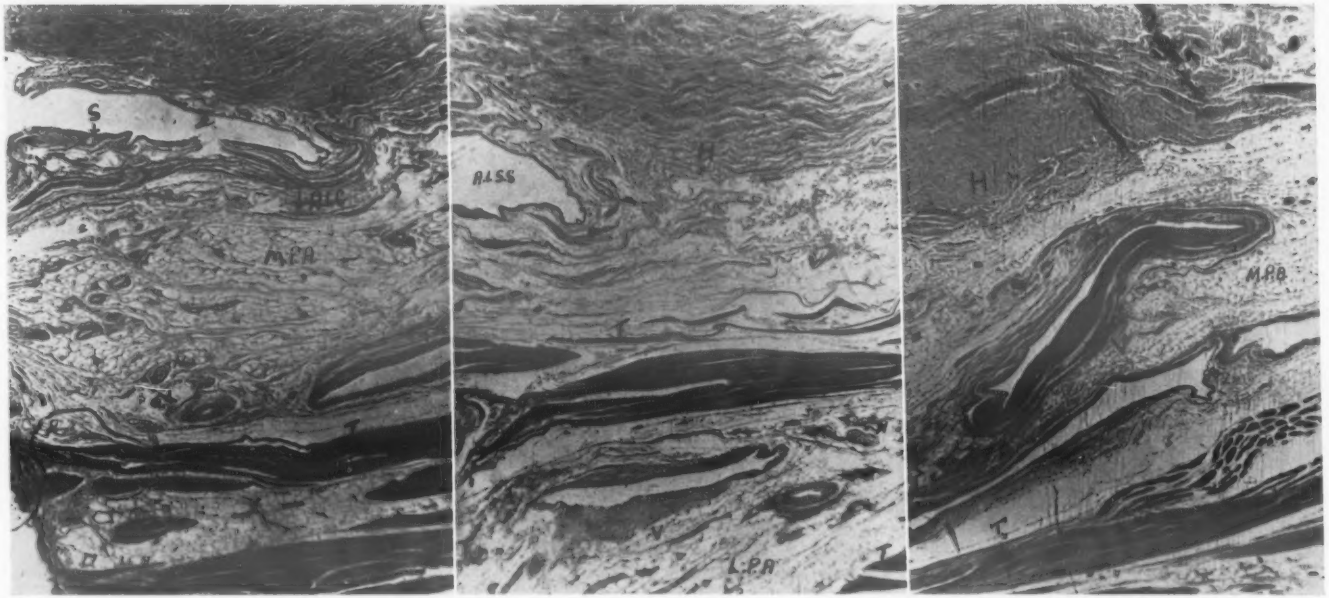


Fig. 10. Fig. 11. Fig. 12.

Fig. 10.—The medial pterygocondylar area (*M.P.A.*). *S*, Synovial membrane. *H*, Hela of the pes menisci. *AIC*, Anterior inferior capsule. *T*, Tendon of the lateral pterygoid muscle.

Fig. 11.—The lateral pterygocondylar area (*L.P.A.*). *A.I.S.S.*, Anterior inferior synovial space. *H*, Hela of the pes menisci. *T*, Tendon of the lateral pterygoid. *N*, Nerve. *V*, Vein.

Fig. 12.—A vein exhibiting plastic thrombophlebitis in the pterygocondylar area.

thrombophlebitis consistent with its being subject to intermittent pressure. It is located in the inferior anterior part of the pterygocondylar area and is in close proximity to the hela of the pes menisci, which forms one side (superior) of the area, and fibers of the lateral pterygoid, which forms the other side of this area (inferior) (Fig. 12).

An account of the vascular anatomy of this pterygocondylar area is now being prepared, but it may be stated here that the reflex symptoms arising from compression in this area probably have to do with vasomotor control.

Roentgenographic diagnosis, as stated previously, is largely a matter of experience and has to be considered in relation to clinical history. Prentiss,[14] Summa,[15] and others have pointed out that in arthrosis the disc becomes thinned and the condyle more intruded in the fossa. It is our belief that such changes occur in the temporomandibular articulation of edentulous patients who fail to have their dentures adjusted concomitant with slow, progressive alveolar ridge absorption and that vascular damage is associated with degenerative changes in the disc. As mentioned previously, Martin[8] pointed out the relative disc space proportions in cadavers with their teeth in normal occlusion. Nearly identical results were obtained by Craddock[16] on roentgenographic analysis. Craddock had the impression that patients with steeply inclined fossae, deep anterior overbite, and Class II malocclusions are predisposed to temporomandibular joint dysfunction. We believe that this concept can be supported on anatomic grounds. Craddock further points out that an abnormally large articular space can be indicative of joint dysfunction and refers to an earlier report by Connors.[17] One of our patients, with whom we experienced great difficulty in treatment, had such a disc space. In our experience the decrement of the disc space, at least in the edentulous patient, appears to be in the direction of facet-forming pressure as outlined by Murphy.[18]

DISCUSSION

A demonstration of plastic thrombophlebitis in this area may not be significant in itself. Nevertheless, we feel that, taken into consideration with the clinical history and demonstrable relationships of the condylar head impinging on the pars gracilis in the articular fossa, it is not coincidental. Furthermore, it adequately explains reflex vascular disturbances so common in our experience in edentulous patients with loss of vertical dimension, the neurones of the afferent limb of the sympathetic reflex are being located in the spinal ganglia of T1 to T4, and the efferent outflow being via the cervical sympathetic trunk with, in certain cases, more caudal components of the thoracolumbar sympathetic trunk being involved. It is only on the basis of the diffuseness of sympathetic reflex arcs that we can account for the variability of the symptoms.

SUMMARY

1. Clinical cases of edentulous patients with decrement of disc space in the occlusal position have been described.

2. An area called the pterygocondylar area has been described.

3. The implication of this area in joint malfunction has been postulated.

4. Plastic thrombophlebitis in this area has been demonstrated.

5. It has been suggested that the variability of symptoms can be accounted for by the properties of sympathetic reflex arcs.

We would like to thank Dr. C. Graham, pathologist of the Royal North Shore Hospital, for examining and commenting on the pathologic sections. We would further like to thank the staff of the Anatomy Department, University of Sydney, especially Mr. B. Lockett, for help in preparing the histologic sections. Thanks are also due Professor A. J. Arnott, Dean of the Faculty of Dentistry, and Professor N. McIntosh, Challis Professor of Anatomy, University of Sydney, for their help and encouragement in these investigations. We are indebted to Mr. Larnach of Sydney University and Miss Simpson of the Royal North Shore Hospital for the photography and to Mrs. Fraser, Almoner Department, Royal North Shore Hospital, for typing the manuscript.

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INTRAORAL APPROACH TO INFRATEMPORAL FOSSA

Reports of Two Cases

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Houston, Texas

With World War II there came an increasing demand for proper management of foreign bodies in the maxillofacial region. The indiscriminate sale of firearms, knives, and other weapons of violence within our own country has transferred these injuries from battlefield hospitals and placed them in our urban medical centers. The problem of intraoral gunshot wounds has not been uncommon.

The removal of foreign bodies within the maxillofacial region is often a difficult surgical procedure. This is primarily due to the distribution of the fifth and seventh cranial nerves and the abundant blood supply. An extraoral approach to this area will always involve close approximation to these nerves, and the danger of facial nerve injury is ever present. An approach via the oral cavity has been used by oral surgeons for the management of many of these maxillofacial problems. This technique has been fostered by the oral surgeon's familiarity with the mouth and associated structures and by the frequency with which he operates in this area.

In this article, we shall describe a method for the intraoral removal of foreign bodies (in this case, bullets) from the infratemporal region of the mandible. This approach can be used successfully for the removal of foreign bodies of various types, such as broken syringe needles, knife blades, glass, etc. Two case reports in which this technique was used will be included along with illustrations that show the regional anatomy.

ANATOMY

The infratemporal fossa is the open space behind the maxilla. It communicates with the temporal fossa through a wide opening under cover of the zygomatic arch. Its anterior wall is the posterior surface of the maxilla, ending inferiorly in the maxillary tuberosity; the roof is chiefly the infratemporal surface of the greater wing of the sphenoid; the medial wall is formed by the lateral pterygoid plate and a narrow strip of the tubercle of the palatine bone; and the lateral boundary is formed by the coronoid process and ramus of the mandible.[1]

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The essential anatomy of the infratemporal fossa is shown in Fig. 1. Of prime importance is the internal maxillary artery. A laceration of this vessel will result in profuse hemorrhage, the treatment for which is packing or ligation. The internal maxillary artery, the larger of the two terminal branches of the external carotid artery, arises at the neck of the mandible

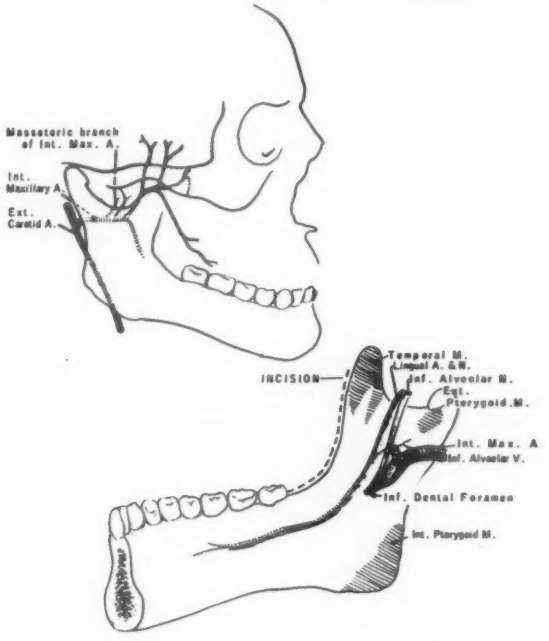


Fig. 1.—Essential anatomy of infratemporal fossa.

and is divided into three parts by the lateral pterygoid muscle. The branches of the first part pass through foramina or canals. They are the deep auricular artery to the external auditory meatus, the anterior tympanic artery to the tympanum, the middle and accessory meningeal arteries to the cranial cavity, and the inferior alveolar artery to the teeth. The branches of the second part supply an artery to each of the following muscles: masseter, temporalis, lateral and medial pterygoid, and buccinator. The branches of the third part arise from the end of the artery within the pterygopalatine fossa. They are the posterior superior alveolar artery, the infraorbital artery, the greater palatine artery, the artery of the pterygoid canal, and the pharyngeal artery.

The area medial to the ramus of the mandible also contains the inferior alveolar and lingual nerves. The inferior alveolar nerve descends behind and lateral to the lingual nerve between the two pterygoid muscles. It winds around the lower border of the lateral pterygoid muscle, which separates the alveolar nerve from the mandibular ramus, and turns out and down to reach

the inner surface of the mandible at the mandibular foramen which it enters. It is important to stress the fact that the inferior alveolar nerve has no contact with the mandible above the entrance to the mandibular canal.[2]

The lingual nerve descends between the pterygoid muscles after its separation from the inferior alveolar nerve and is closely united with this nerve, lying anterior and slightly medial to it. At the lower border of the lateral pterygoid muscle the lingual nerve receives the chorda tympani which contains visceral efferent and taste fibers from the facial nerve. Below the lateral pterygoid muscle the lingual nerve follows the lateral surface of the medial pterygoid muscle, coursing down and laterally.

The inferior alveolar nerve and its branches supply sensory innervation for the teeth, the buccal and labial gingivae, the lower lip, and the skin of the chin. The lingual nerve supplies the anterior two-thirds of the tongue, the lingual gingiva, and the floor of the mouth.[2]

TECHNIQUE

After satisfactory anesthesia is obtained, an incision is made 1 cm. posterior to the retromolar pad through the mucoperiosteum. This incision is carried along the ascending ramus to the superior third of the anterior border of the coronoid process. With periosteal elevators, the mucoperiosteum is elevated from both the medial and lateral aspects of the coronoid process and the superior third of the ascending ramus of the mandible. This reflection exposes the tendinous insertion of the temporalis muscle into the coronoid process and the anterior border of the ramus. By blunt dissection of the mucoperiosteum on the medial aspect, the area of the sigmoid notch is visualized. Extreme care must be taken not to lacerate the internal maxillary artery and especially its masseteric branches which course through the sigmoid notch. Caution also must be exercised not to injure the inferior alveolar and lingual nerves, whose courses have already been described. This approach now provides access to the infratemporal fossa, which can be explored by blunt instrumentation for foreign bodies.

In addition to the information that radiographs will provide, sometimes foreign bodies may be localized by palpation, by both objective resistance transmitted to the finger and subjective pain produced by certain manipulations while the patient is being examined.[3]

Hemorrhage is usually caused by laceration of the branches of the pterygoid plexus and may be controlled by gentle packing with Gelfoam. After the foreign body is removed, the incision is closed with 0000 chromic sutures. If hemorrhage is copious, a small Penrose drain may be inserted at the most inferior part of the incision. This may be removed in twenty-four hours.

CASE REPORTS

CASE 1.—A 34-year-old Latin man in acute distress was admitted to the surgical service of the Veterans Administration Hospital in Houston, Texas, on Dec. 3, 1956, with multiple gunshot wounds of the head and body. There was a bullet wound over the left eye, with the

route of exit not seen. There was periorbital extravasation of blood about the left eye. The multiple gunshot wounds of the body, other than the wound over the left eye, were treated by the surgical service, and on Dec. 17, 1956, the patient was transferred to oral surgery. Roentgenograms taken in the dental clinic revealed a bullet lodged in the infratemporal fossa (Fig. 2, *A* and *B*).

On Jan. 2, 1957, following induction of general anesthesia, the patient was scrubbed and draped in the usual manner for an intraoral procedure. An incision was made posterior to the retromolar pad through the mucoperiosteum, extending along the anterior border of the ascending ramus to the midportion of the coronoid process. Because of the presence of the masseteric artery, vein, and nerve, special attention was given to the medial reflection as the area of the sigmoid notch was approached. After the reflection was accomplished, blunt probing of the area revealed a bullet lodged near the sigmoid notch. The bullet was removed with a considerable amount of hemorrhage from minor branches of the pterygoid plexus. The bleeding was controlled by gentle packing with Gelfoam, and the incision was closed with interrupted 0000 chromic sutures. The patient had an uneventful postoperative course and was discharged on Jan. 10, 1957.

CASE 2.—A 38-year-old Negro man was seen in the emergency room of Jefferson Davis Hospital on June 9, 1957. History revealed that the patient had been involved in an argument and had received a gunshot wound. The bullet entered below the right angle of the mandible and went through the right ramus, lodging medial to it. The x-ray report was as follows: "There is a gunshot wound of the right mandible with a comminuted fracture of the ramus and a heavy metal mass, representing the bullet, just medial to this. Location of this would be immediately below and also above the hard palate, probably above the tonsillar fossae." (Fig. 3, *A* and *B*.)

The patient was taken to the operating room and, under local anesthesia, Stout's intermaxillary wiring was applied to the teeth. Good occlusion was obtained with the closed reduction. The patient was followed in the outpatient clinic for routine postoperative treatment. The healing of the fracture was uneventful, but the patient complained that he had difficulty in opening his mouth wide. It was decided to remove the bullet to see whether or not preinjury function could be restored.

On Aug. 28, 1957, the patient was premedicated with $\frac{1}{6}$ gr. morphine and $\frac{1}{150}$ gr. atropine and taken to the operating room. After satisfactory general anesthesia had been obtained, the patient was scrubbed and draped in the usual manner for an intraoral operation. The bullet was then removed by the intraoral surgical method described. There was little hemorrhage during the operation. The incision was closed with 0000 chromic sutures. The patient left the operating room in good condition. Very little swelling was evident the first postoperative day. On the third postoperative day the patient was discharged. On return visits to the dental clinic the patient said that he could open his mouth "easier and wider." The patient was discharged on Sept. 26, 1957.

COMMENT

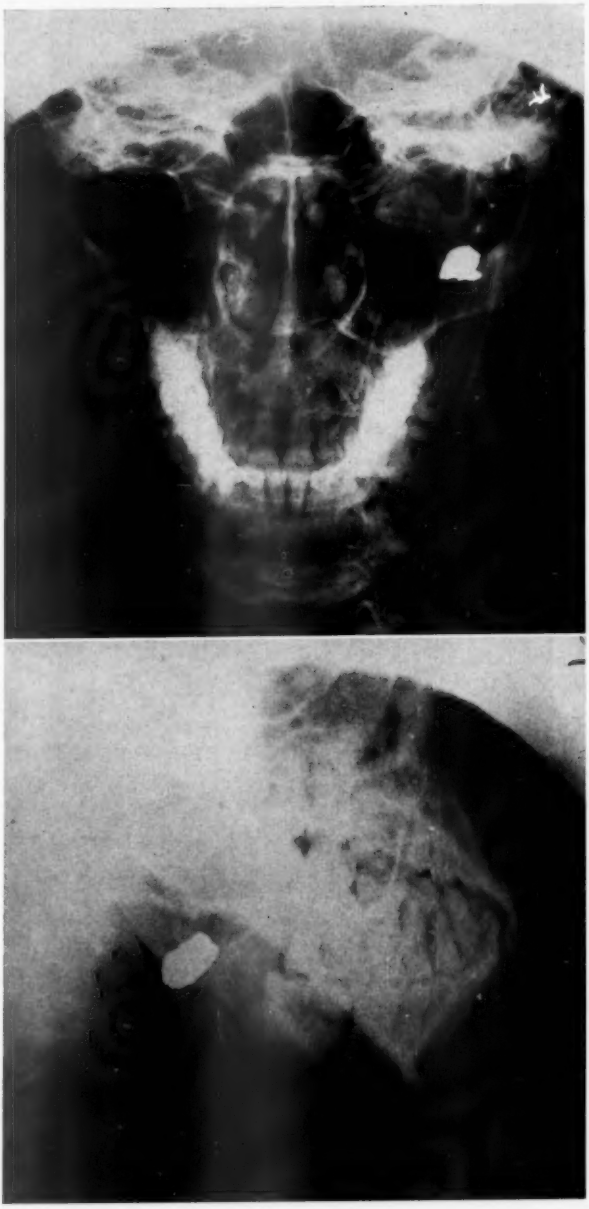
There are several advantages to the intraoral surgical approach for removal of foreign bodies:

1. *Better access.* Direct approach to the medial surface of the ascending ramus is possible.

2. *Local anesthesia.* In many instances it is possible to use local anesthesia rather than subject the patient to a general anesthetic agent.

3. *Rapidity of healing.* Because of the extreme vascularity of the oral tissue, intraoral operative wounds heal much faster than extraoral ones and there is less chance of postoperative infection. This is due in part to autoimmunologic factors and to the bacteriostatic action of saliva.

A.



B.

Fig. 2.

A.

B.

Fig. 3.

4. *Cosmetic effect.* Facial scarring is eliminated.

5. *Less chance of injury to branches of the facial nerve.* The major branches of the seventh nerve lie lateral to the field of this surgical approach.

DISCUSSION

The problem of whether to remove foreign bodies routinely or to leave them until definite symptoms occur as a result of their presence is usually discussed. Although questions concerning the nature and extent of immediate damage to muscle fibers resulting from temporary cavity formation remain unanswered, subsequent degenerative changes are known to occur in traumatized muscle and other soft tissues. Such changes are abundantly described in the literature on pathology.[4]

When foreign bodies remain buried in the tissues for long periods of time in close proximity to blood vessels, there is a possibility of erosion taking place with subsequent hemorrhage and hematoma. Inflammatory changes involving lymphatic structures have produced lymphostasis, and enlargements of the lymph nodes resembling those seen in Hodgkin's disease and tuberculosis have been described.[5] In addition, fibrosis associated with the walling-off of foreign objects may encompass a nerve resulting in dysfunction or irritation.

Certainly, removal is indicated when there is limited motion of the jaw due to muscular or bony impingement, when there is pain, or excessive irritation to the involved tissues, or when a low-grade chronic infection is present and all possible foci of infection must be eliminated. Dalitsch[6] says that foreign bodies predispose to infection and their removal is a safeguard against such infection.

When removal would necessitate an unwarranted danger to the patient's life, an operation is contraindicated, as foreign bodies lodged within the tissues may not cause an active infection. Throndson[7] states that when a foreign material is sterile, essentially insoluble, and only slightly reactive to tissue fluids, the body tolerates it well. However, the absence of definite symptoms does not necessarily mean that a chronic infection has not been present and has subsided and that it will not activate itself in the future.

It is our opinion that a foreign body of significant size should be removed prophylactically from the maxillofacial region of young persons when their health permits; in persons of advanced years, caution should be exercised until definite symptoms appear. The removal should be accomplished as soon as possible following injury.

CONCLUSIONS

1. The incidence of maxillofacial injuries has increased during the last generation because of war and because of the indiscriminate sale of firearms in our country.

2. An intraoral surgical approach to the infratemporal fossa for removal of foreign bodies has been described.

3. This technique offers the advantages of local anesthesia, better access, rapid healing, less chance of postoperative infection, better cosmetic effect, and less chance of injury to branches of the facial nerve.

4. Whenever possible, all foreign bodies of significant size should be removed from the maxillofacial region.

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RECURRENT ODONTOGENIC CYSTS

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THE greatest number of cysts of the jawbones are of odontogenic origin. Two groups are classified: (1) the follicular cyst associated with the crown of the tooth and (2) the periodontal cyst associated with the root of the tooth. These are benign lesions; however, recurrence will follow incomplete removal.

The following case demonstrates recurrences of both types of cysts in the same patient.

CASE REPORT

History of Present Illness: In December, 1949, at the age of 18 years, patient A. S. G. had an impacted lower left third molar and an associated follicular cyst removed. In September, 1953, it was discovered that the cyst had recurred. The area was again entered intraorally, and a cystectomy was performed. At this time the lower left second molar was removed and a radicular cyst associated with the tooth was enucleated. From information furnished by the patient, it was inferred that in both operations on the follicular cyst the cavity was packed open.

Radiographic examinations in May, 1954 (Fig. 1), and November, 1954 (Fig. 2), disclosed recurrences of both lesions with evidence of rapid proliferative activity.

The patient was referred to the oral surgery service at Rodriguez Army Hospital for evaluation and treatment of his condition.

Past History and Family History: A history of familial tendency toward cyst formation could not be elicited. All other history was noncontributory.

Examination: The lower left second and third molars were missing. The mucosa in the left retromolar triangle was bound by scar tissue to the underlying periosteum as a result of the previous surgical procedures.

On the posteroanterior radiograph of the mandible, both lesions appeared to occupy the medullary portion of the bone between the lateral and medial cortices. A radiographic skeletal survey disclosed no other abnormalities.

General Physical Findings: The patient was a well-developed and well-nourished 24-year-old white man. His temperature was 98.2° F., and his blood pressure was 108/68. He was examined by a physician of the medical service, who reported no significant physical defects.

Laboratory Examination: The hemogram, routine flocculation, and urine examinations were negative. Serum calcium, serum inorganic phosphorus, and alkaline phosphatase were within normal limits.

Treatment and Course: On Dec. 11, 1954, the mouth was treated with tincture of Zephiran chloride 1:1,000. Under inferior alveolar block anesthesia (2 per cent lidocaine

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Fig. 1.

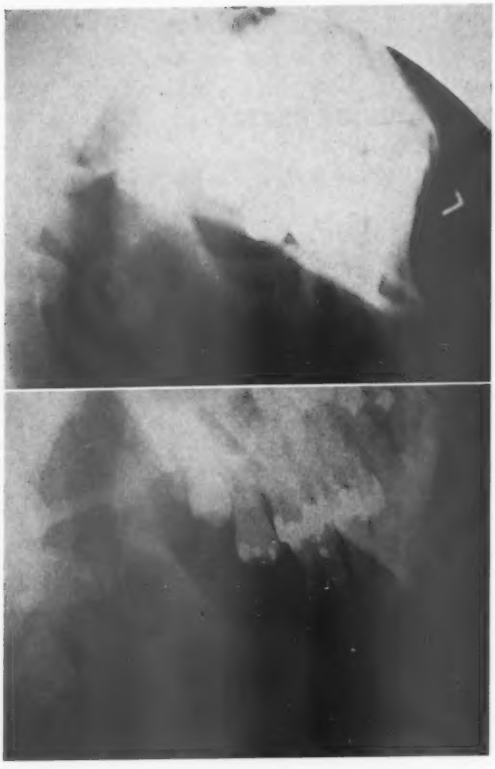

Fig. 2.

Fig. 1.—Residual cysts seven months preoperatively.
Fig. 2.—Preoperative radiograph of residual cysts.

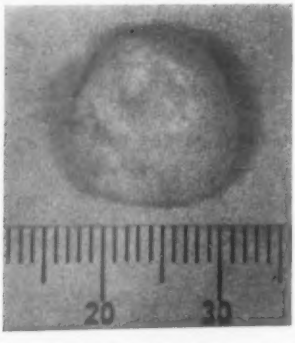

Fig. 3.—Cyst enucleated from body of mandible.

with epinephrine 1:50,000), an incision through mucoperiosteum was made, extending from the left retromolar triangle over the crest of the alveolar ridge to the distal surface of the first molar. The incision was then carried obliquely downward and forward to the buccal sulcus. The mucoperiosteal flap was elevated and retracted laterally. The lateral surface of the body of the mandible was exposed, and several drill holes were cut through the cortical bone to circumscribe the lesion. Cuts were made to join the drill holes, and the bone overlying the lesion was removed. A dull gray cyst wall was exposed. The cyst was completely enucleated by means of angled curettes (Fig. 3). The area was débrided, and the cavity was filed smooth and permitted to fill with blood. The mucoperiosteal flap was approximated with 0000 silk interrupted sutures.

The patient had received 600,000 units of procaine penicillin intramuscularly daily for two days preoperatively as a prophylactic measure. This was continued for three days postoperatively. Other postoperative treatment consisted of an ice pack to the left side of the face for twenty minutes every hour in the immediate postoperative period and hot saline irrigations intraorally every hour starting the following day.

The postoperative course was mild. There was a traumatic edema which subsided completely by the third day following surgery. The sutures were removed on December 15.

The pathologic report was as follows:

Gross: The specimen consists of a white cyst measuring 1.6 cm. in diameter. The wall is very thin and contains a little soft, whitish material.

Microscopic: These sections reveal portions of a cyst with a thin wall lined by stratified squamous epithelium. A few small foci of chronic inflammation (and one with foreign material) are noted in the wall.

Diagnosis: Radicular cyst.

On Feb. 15, 1955, the patient was admitted to the oral surgery service at Rodriguez Army Hospital for removal of the cyst in the ramus of the left mandible. Routine laboratory studies, chest x-ray, and physical examination were again negative.

The patient received 600,000 units of procaine penicillin intramuscularly once daily as a prophylactic measure. He was given 100 mg. of Nembutal sodium that night. One hour preoperatively, 100 mg. of Demerol hydrochloride and 0.4 mg. of scopolamine were administered by hypodermic injection.

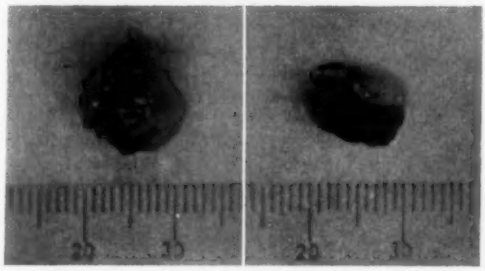

Fig. 4. Fig. 5.

Fig. 4.—Hyperplastic submandibular lymph node
Fig. 5.—Cyst enucleated from ramus of mandible.

On February 16, under intravenous thiopental sodium and endotracheal nitrous oxide-oxygen anesthesia, the face and neck were prepared with a synthetic detergent containing hexachlorophene and the patient was draped in a sterile manner. An incision 5 cm. in length through skin, subcutaneous tissue, and platysma muscle was made 1.5 cm. below the lower border of the mandible from the angle of the mandible anteriorly, following the natural lines of tension. Bleeding vessels were clamped with fine-pointed hemostats and tied with 0000 plain catgut. By sharp and blunt dissection, the lower border of the

mandible was exposed. A hyperplastic lymph node in the surgical field was excised (Fig. 4). The periosteum at the lower border of the mandible was incised and elevated. The flap containing the masseter muscle, parotid gland, and facial nerve was retracted to expose the lateral surface of the mandibular ramus. A fenestration was produced in the bone overlying the cyst by means of a Henahan burr in the dental engine and Kerrison upcutting forceps. In this procedure the cyst was perforated, and about 1 c.c. of viscous white fluid escaped. The collapsed cyst was grasped with a hemostat and easily removed from its crypt (Fig. 5). The bone cavity was flushed with warm saline, inspected, and found to be free of any residual material. Gelatin sponge was placed in the bone cavity to fill the defect.

Closure was done in layers. The masseter muscle was sutured to periosteum at the angle and lower border of the mandible by means of 00 chromic catgut. Platysma muscle was approximated by means of 0000 chromic catgut, and final skin closure was obtained with 36 gauge subcuticular wire. A light pressure dressing was applied.

The pharynx was aspirated, and the patient was extubated and returned to the ward reacting and in good condition. An infusion of 1,000 c.c. of 5 per cent glucose in water, started in the operating room, was permitted to continue until the patient had received the entire volume.

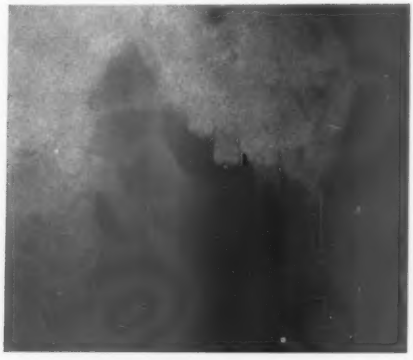

Fig. 6.—Radiograph taken seven months postoperatively. The cystic areas are traversed by trabecular markings.

Postoperatively, the patient received 600,000 units of procaine penicillin intramuscularly once daily for five days. An ice pack was placed to the left submandibular area for twenty minutes every hour in the immediate postoperative period.

On February 17 the patient presented a moderate swelling of the left submandibular area. Slight trismus was present. There was no disturbance of motor control to the lower lip.

On February 21 the subcuticular wire was removed, and the patient was discharged to duty.

The pathologic report was as follows:

Gross: Specimen A consists of a small cyst with collapsed walls. Specimen B consists of a lymph node measuring 1.5 cm. in greatest dimension.

Microscopic: Slide A shows a cyst lined by squamous epithelium and having some chronic inflammatory cells in the wall. Slide B reveals a lymph node showing some lymphoid hyperplasia with obscuring of many sinuses.

Diagnosis: Residual follicular cyst of mandible. Chronic lymphadenitis.

Result: Radiographic examination seven months after the second operation disclosed a trabecular pattern filling in the bone defect. The outlines of the previously cystic areas were less distinct (Fig. 6).

The scar produced as a result of the surgical procedure was not objectionable cosmetically (Fig. 7).

Fig. 7.—Scar produced at site of incision.

COMMENT

The decision to remove the cyst in the ramus of the mandible through an extraoral approach was based on several factors. The area had been entered twice intraorally, with recurrence after each operation. The cicatrix formed as a result of these operations would have made access to the lesion difficult to obtain. Furthermore, since ameloblastoma may occur in relation to the follicular cyst,[1] it was considered advisable to use an approach that would provide the best possible access so that complete enucleation would be ensured.

DISCUSSION

A follicular cyst is formed following retrograde changes in the stellate reticulum accompanied by an increase in the fluid content of the developing enamel organ by transudation from the perifollicular space. The cyst lining is derived from the outer enamel epithelium, and the cyst wall from the perifollicular connective tissue.

The periodontal cyst is formed by the proliferation of epithelium derived from the debris of Malassez, remnants of Hertwig's root sheath. As the cells proliferate, those in the center become farther removed from their blood supply. These cells die, and a central cavity is created. This may be considered the earliest sign of a periodontal cyst.

The cells that line a follicular cyst have the same origin and appear to be similar to those that line a periodontal cyst; yet, ameloblastoma has been found in the former but not in the latter. A possible explanation for this phenomenon lies not in these cells themselves but, rather, in the influence of neighboring cells whose presence is necessary for producing change. The cells that line the follicular cyst are associated in odontogenesis with the cells of the stellate reticulum and stratum intermedium. The cells that line the periodontal cyst are derived from remnants of Hertwig's sheath, a structure that contains only inner and outer epithelium, deficient in stellate reticulum and stratum intermedium. It is not illogical to assume that follicular cyst epithelium, having been stimulated by stellate reticulum and stratum intermedium, could develop in its predestined pattern toward amelogenesis but, lacking the inductive influence of mesoderm, would not produce enamel and would continue to proliferate and form an ameloblastoma. In other instances, in which the inductive influence of mesoderm is present, an odontoma is formed. Bearing this in mind, one must consider the neoplastic potentialities of follicular cysts, and the surgical approach therefore should be directed toward complete enucleation.

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TRAUMATIC MANDIBULAR BONE CYST INVOLVED IN LINE OF FRACTURE

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TRAUMATIC mandibular bone cysts, or hemorrhagic cysts, are usually symptomless cavities of debatable etiology. Olech, Sicher, and Weinmann,[6] reviewing twenty cases and presenting eight more, found a history of trauma in twenty-one out of the twenty-eight cases. Of twenty-four cases reported since,[1-5, 7, 9] there was a definite history of trauma in eleven. (The etiology and pathology are fully discussed in a symposium entitled "Solitary Bone Cysts of the Mandible," published in the September, 1955, issue of this JOURNAL. The average age of the patients was 19 years with an age range of 11 to 39 years.) The case described here, unlike those above, was discovered because of its involvement in a fracture.

CASE HISTORY

The patient, a 19-year-old white man, employed on kitchen duties, slipped on March 14, 1956, and struck his face against the bacon slicer. He did not lose consciousness, and little pain was felt at the time. The next morning he had swelling on the left side of his mandible and, to a greater degree, on the right. He then reported to his unit dental officer, who diagnosed a fracture through a cystic cavity in the right mandible. The patient was transferred to the hospital on March 15, 1956.

On examination, he presented with swelling over the left angle and right body of the mandible. Slight anterior open-bite, limitation of opening to one fingerbreadth, and an occlusal step in the $\overline{45}|$ region where the gum was lacerated were noted. There was no paraesthesia of the lips; no bony expansion of the mandible could be felt; and no history of previous trauma in this region could be elicited. No carious teeth were related to the cavity, and all the mandibular teeth on the right side gave vital responses to electric pulp testing.

Roentgenographic Examination.—Radiographs (Figs. 1, 2, and 4) showed a linear fracture with no displacement from the distal aspect of $|\overline{8}$ to the lower border; a linear fracture through the $\overline{5}|$ socket, with slight displacement; and a large, well-defined, radiolucent area in the body of the mandible from $\overline{3}|$ to $\overline{7}|$, extending to the lower border and up between $\overline{5}|$ and $\overline{6}|$, the lamina dura of all teeth appearing intact. A posteroanterior view (Fig. 3) showed no expansion. Radiographs of the long bones, pelvis, and skull were normal.

Laboratory Investigations.—Blood alkaline and acid phosphatases were within normal limits, as was the serum calcium. Blood cytology was normal.

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Diagnosis.—Bilateral fracture of the mandible, complicated by a cyst on the right side. On the basis of clinical and radiographic examination, it was thought likely, that this was a traumatic mandibular bone cyst which had been present without symptoms for some time. It was obvious that a firm diagnosis was impossible without a biopsy.

Treatment.—On March 15, 1956, penicillin cover (600,000 units procaine penicillin daily) was instituted, and a headcap and elastic support were fitted while cap splints were being made. On March 16 sectional cap splints were cemented. The following day elastic traction was instituted. On March 19 elastics were replaced by wires and a locating bar was fitted between sectional splints.

Fig. 1.

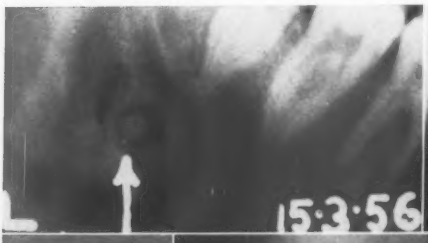
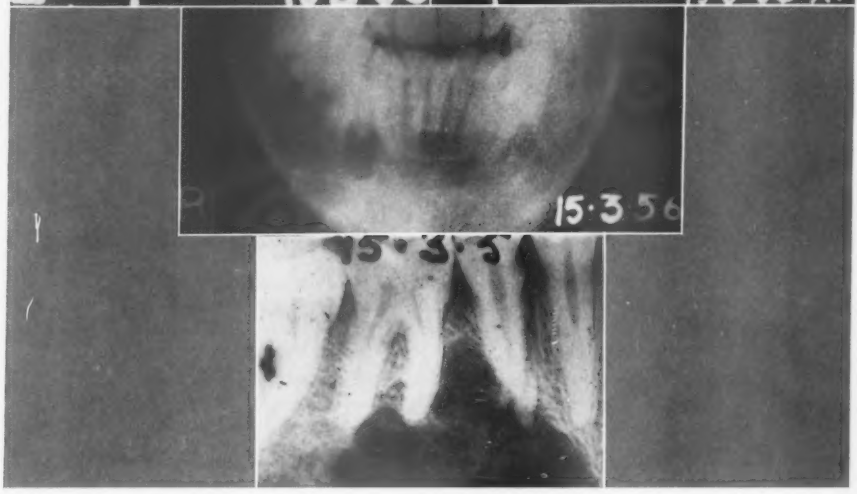


Fig. 2.

Fig. 3.

Fig. 4.



Under nasotracheal anesthesia, a biopsy was performed on March 20, 1956. An incision was made in the buccal sulcus in the $\overline{654}$ | region; the mucoperiosteum was reflected, and a window was cut (well below the line of incision) in the outer plate at the line of fracture. Only serous fluid, which did not appear to be under pressure, was evacuated from the cavity. The roots of $\overline{5}$ | and $\overline{6}$ | could be seen protruding into the cavity, and exploration failed to reveal a lining.

No further operative procedure was deemed necessary. The mucoperiosteum was replaced and sutured. The portion of outer plate removed was sent to the pathology laboratory for sectioning.

By April 4 progress had been uneventful. There was a sound scar in the sulcus, and the penicillin cover was withdrawn (total administered, 12 mega-units).

On April 23 the intermaxillary wires were removed and the fracture was tested. This showed sound clinical union, but it was decided to retain the locating bar on the lower splint across the fracture line.

On April 27 the patient was discharged to ten days' sick leave.

When the patient was seen on May 8 it was noted that sound clinical union had been maintained. The splints were removed. Teeth $\overline{76543}$ | still gave vital responses. The slice in $\overline{5}$ | was restored with an amalgam filling.

On May 15, under nasotracheal anesthesia, | $\overline{8}$ was removed. An intraoral radiograph (Fig. 5) taken three days later revealed increased opacity of cyst, suggesting bone regeneration.

Fig. 5.

The patient was discharged to duty on May 24, 1956.

Clinical Reviews.—When the patient was seen on Aug. 24, 1956, he had no signs or symptoms. An intraoral radiograph was taken at that time (Fig. 6).

On Nov. 16, 1956, the patient was clinically and radiographically normal. X-ray films (Figs. 7, 8, and 9) showed that the cysts had ossified well. Teeth $\overline{76543}$ | gave vital responses and were normal on percussion.

Pathologist's Report.—Sections showed an area of compact bone which had been denuded of periosteum at operation.

The inner surface showed lacunar resorption by osteoclasts and, in addition, there was a thin layer of fibrous tissue with practically no collagen fibers.

There was no epithelial lining present. The appearances were compatible with the diagnosis of "traumatic or hemorrhagic cyst." The microscopic appearance is shown in Figs. 10 and 11.

RÉSUMÉ ON TREATMENT

Various methods of treatment have been described in the literature. One frequently used is surgical exposure and suture of the flap, allowing the cavity to fill with blood clot, as reported by Olech, Sicher, and Weinmann,[6] Jacobs,[5] and Whinery.[9] Partial or complete packing of the cavity leads to healing by granulation, according to Thoma[7] and Fickling.[4] Gelfoam has been used to pack the cavity before suturing of the flap, with good results, as reported by Thoma[7] and Whinery.[9] Boyne[3] used freeze-dried homogenous bone grafts with good results in two cases. Blum[2] described several cases that have regressed without intervention.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

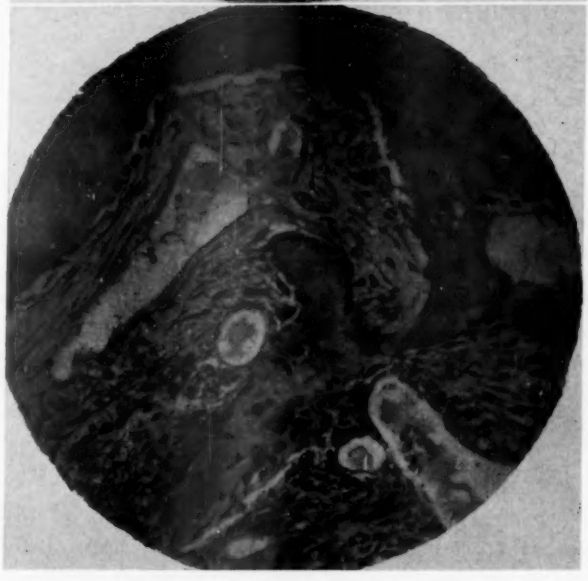


Fig. 10.—Magnification, ×200; reduced ⅙.
Fig. 11.—Magnification, ×800; reduced ⅙.

CONCLUSIONS

Clinically these cysts appear to confine themselves to the destruction of cancellous bone, leaving the lamina dura intact around the teeth and giving an almost characteristic radiographic appearance. In the case described here, simple exposure and aspiration of the contents, coupled with adequate antibiotic cover, resulted in the healing of a traumatic mandibular bone cyst with complete regeneration of bone within six months, despite its involvement in a fracture.

Acknowledgment is made to the Director of Dental Services, Royal Air Force, Air Vice Marshall M. J. Pigott, QHDS, F.D.S., R.C.S., B.D.S., for permission to publish this article.

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EXODONTIA

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THE PROBLEM OF IMPACTED TEETH

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THE removal of impacted teeth is not without problems. Although their removal is a frequent occurrence, it should not be undertaken by the untrained surgeon. Removal of impacted teeth requires training, clinical judgment, and experience if problems are to be avoided. As with any surgical procedure, there may exist a difference of opinion as to why, when, and how impacted teeth should be removed. Each opinion, however is based on sound surgical and physiologic principles.

If one reviews the literature, he will find oral surgeons who believe that impacted teeth should be removed if they cannot erupt or be brought into a normal position in the dental arch. The rationale given for their removal is that (1) impacted teeth cause irregularities and deformities in the dental arch, such as the crowding of anterior teeth, and (2) they may develop cysts, pericoronal infections, and Vincent's infection. Impacted teeth have been described by some authors as the etiological factor in reflex pain, neuralgias, nervous disorders, neuritis, tinnitus, and muscular twitchings.[1-4] Because of this, some believe in prophylactic odontectomy of these teeth. Thoma[5] states that he prefers to remove the third molar when the roots are three-quarters formed. Others believe that lower third molars should be retained until maximum growth of the face and jaws has been attained, which should be between the sixteenth and eighteenth years. The delay in removal will assist vertical development in the premolar and molar regions.

If one had to cite indications as to when impacted teeth should be removed, the following might serve as a guide:

1. When, in the early development, the impacted tooth causes a tilt of the adjacent tooth which may result in a partial or complete impaction of that tooth (Figs. 1 and 2).

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2. When the position of the crown of the impacted tooth is locked against the distal root of the adjacent tooth, which may result in absorption of the root (Fig. 3). Unfortunately, roentgenographic examination may not give a true picture (Fig. 4).

3. When the orthodontist recommends removal of the impacted tooth because of an orthodontic problem (Figs. 5, 6, and 7). There exists a difference of opinion among orthodontists as to the part that

Fig. 1.

Fig. 2.

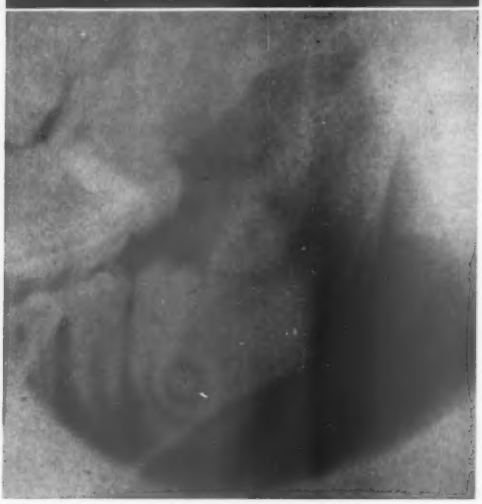
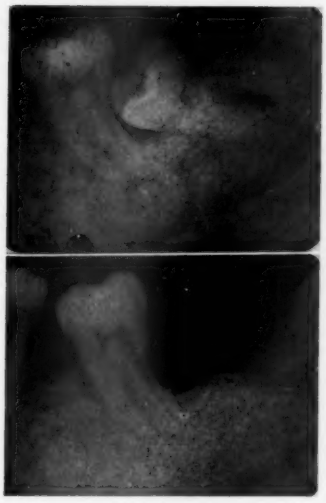


Fig. 1.—Lateral oblique roentgenogram showing impacted third molar causing impaction of second molar.

Fig. 2.—Postoperative roentgenogram showing second molar in position following removal of impacted third molar.

A.



B.

Fig. 3.—Preoperative and postoperative roentgenograms showing absorption of distal root.

A.

B.

Fig. 4.—*A*, Preoperative roentgenogram suggesting possible absorption of distal root. *B*, Postoperative roentgenogram negative for absorption of distal root.

an impacted tooth plays in occlusion or its correction. The surgeon should follow the orthodontist's treatment plan, since the orthodontist is responsible for the results.

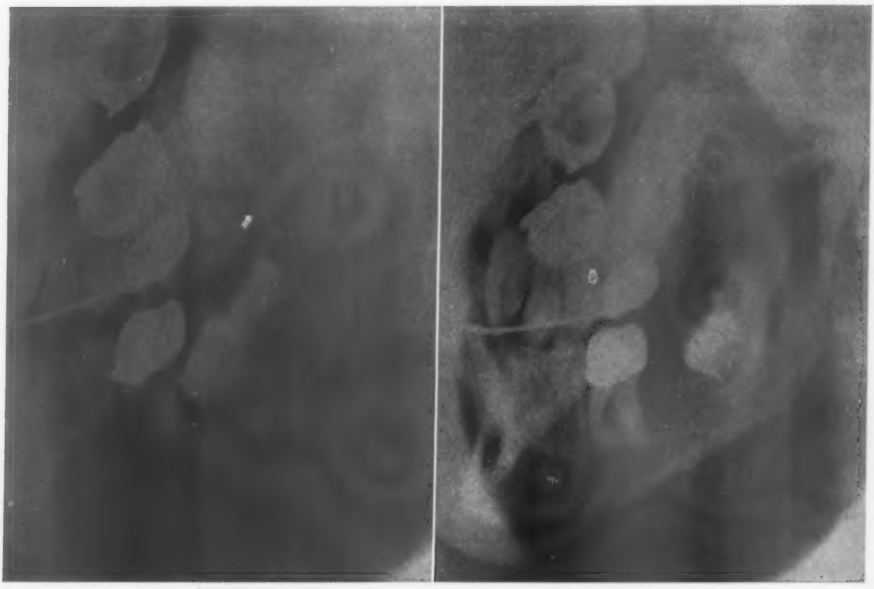


Fig. 5. Fig. 6.

Fig. 5.—Lateral oblique roentgenogram showing impacted second and third molars.
Fig. 6.—Lateral oblique roentgenogram showing removal of impacted second molar.

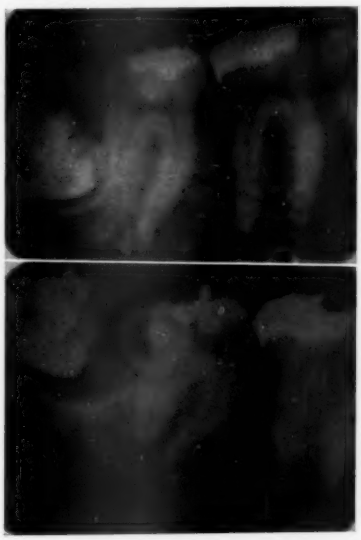
Fig. 7.—Postoperative roentgenogram showing position of third molar following orthodontic treatment.

4. When the tooth is partially impacted it serves no purpose in mastication and it may be responsible for occlusal disturbances. The partially impacted tooth is susceptible to acute or chronic pericoronal infections, or it may be involved in acute Vincent's infection. The follicular sac of the partial impaction may proliferate into cyst formation.

5. When the impacted tooth may be the cause of many disturbing complaints and symptoms, either localized or reflex.

6. When the impacted tooth is in an edentulous tissue-bearing area. The stimulation of the prosthesis, the resorption of the ridge, or the combination of both may result in symptoms of pain, irritation, and even eruption of the tooth.

A.



B.

Fig. 8.—Roentgenogram showing development of dentigerous cyst about impacted third molar, over a period of two years, illustrating the need for periodic roentgenographic examination.

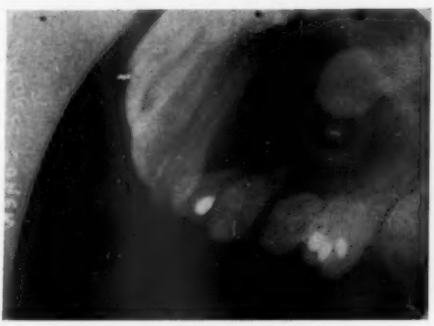


Fig. 9.—Roentgenogram revealing extensive dentigerous cyst about impacted maxillary canine.

Complete bony impactions can be retained when they are free of symptoms and are roentgenographically negative, provided that they are examined periodically (Fig. 8). Dentigerous cyst formation about impacted teeth is not

infrequent and, when formed, these cysts will grow extensively (Fig. 9). Some of these dentigerous cysts (Fig. 10) may be potential ameloblastomas or true ameloblastomas.[6, 7]

The problem of when impacted teeth should be removed is somewhat controversial. Removal of the asymptomatic impacted tooth is an elective surgical procedure. It can be done by whatever operative technique and anesthesia the surgeon prefers. In the presence of acute infection, however, there exists some difference of opinion among oral surgeons as to the time

Fig. 10.—Roentgenogram showing impacted tooth with preoperative diagnosis of dentigerous cyst. Postoperative microscopic examination of tissue revealed ameloblastomatous changes in cyst wall.

for removal. Krogh[8] sees no contraindication to the removal of impacted teeth in the presence of acute infection, provided that proper surgical technique and anesthesia, with antibiotics, are utilized when necessary. Cameron[9] feels that the infection may enter the uninfected bone or migrate into the deeper soft tissues. Winter, Harrigan, and Winter[10] feel that removal is not contraindicated, provided that anesthesia can be obtained without injection into the area. However, they urge caution with the mandibular third molar because of its anatomic relationship to the structures of the throat, floor of the mouth, and neck. In the final analysis, one must agree that each surgical case should be evaluated on its own merits with respect to the time for removal of the acutely infected tooth. The time for surgical intervention, the anesthesia of choice, and the surgical technique must be left to the judgment and experience of the oral surgeon.

The surgical technique for removal of impacted teeth has been thoroughly described in the various texts on oral surgery. However, the basic surgical principles of flap design giving adequate exposure of the operative site with no interference with its vascular supply cannot be ignored. The actual technique may vary with the training and experience of the surgeon.

Fig. 11.

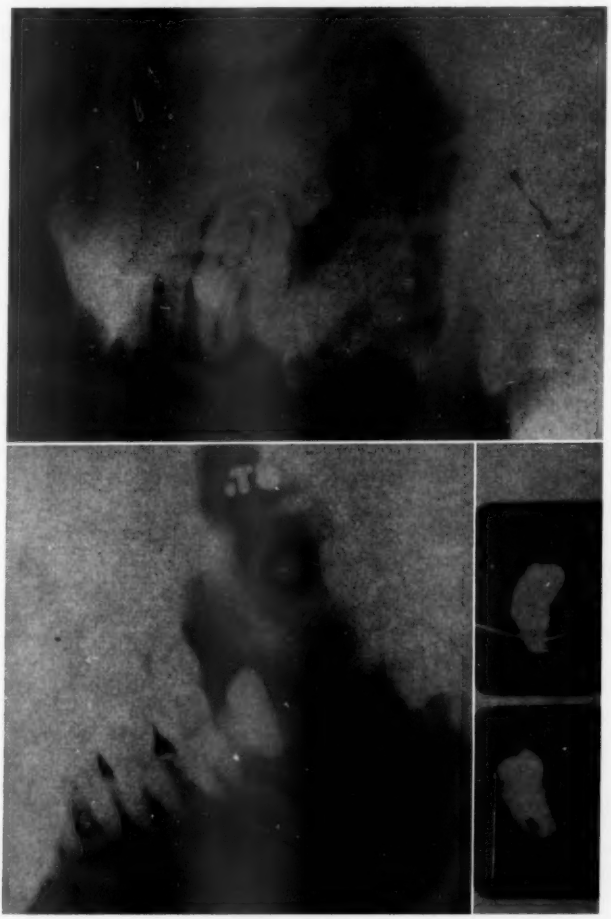


Fig. 12.

Fig. 11.—Roentgenogram showing impacted tooth anatomically related to inferior alveolar canal. Postoperative paresthesia should be anticipated.

Fig. 12.—Roentgenogram revealing impacted mandibular third molar. The root of the tooth made up the walls of the inferior alveolar canal.

The removal of the impacted tooth by the untrained surgeon is not without its complications. Inadequate exposure, blind probing, and undue force

applied to the tooth may displace it into the adjacent structures, fracture the bone, or cause paresthesia.

Impacted teeth may be anatomically related to the inferior alveolar canal. They may be in close proximity or make up the walls of the canal. Surgical trauma, be it normal or excessive, may cause paresthesia. Those impactions which are closely related to the walls of the inferior alveolar canal will, of necessity, cause paresthesia (Figs. 11 and 12). When paresthesia is anticipated preoperatively, the patient should be so advised.

Impacted teeth, because of their anatomic relationship to the adjacent structures, may also be displaced during removal. Their position in the soft or hard tissues of the oral cavity may be the result of accident, either avoidable or unavoidable. When the teeth are surgically displaced in the adjacent tissues, they become foreign bodies and should be removed.

CASE REPORTS

CASE 1.—A 17-year-old white boy was referred by his dentist with a chief complaint of pain and swelling of the left face of one day's duration.

History of the present illness revealed that on the preceding day the patient's dentist attempted to remove an impacted maxillary first molar. During the surgical procedure, the tooth was forced into the maxillary sinus.

Clinical examination revealed swelling of the left face, extending from the infraorbital ridge to the zygoma. There was crusted blood at the left naris. An intraoral examination revealed lacerations of the buccal and palatal mucous membrane in the first molar region. The lacerations extended from the mucobuccal fold to the crest of the ridge. A silk suture had been placed buccally to hold and adapt the mucous membrane; however, there was a direct oroantral communication.

Roentgenographic examination showed the tooth in the maxillary sinus (Figs. 13 and 14).

The patient's medical history was essentially negative.

Because of the lacerations of the mucous membrane, it was decided that surgery should be postponed until healing took place, since all available mucous membrane would be needed to obtain a primary closure of the oroantral communication. The patient was placed on antibiotic therapy and sedation.

Three days following the initial visit, the suture was removed. The mucous membrane had healed satisfactorily. A mucous discharge was noted at the oroantral communication.

The following week it was decided that surgery could now be performed, since the soft tissue was in a satisfactory state of repair. Under local anesthesia consisting of an infraorbital and a posterior superior alveolar nerve block and infiltration, both buccally and palatally, a modified Caldwell-Luc incision was made from the canine to include the original incision and extend along the crest of the ridge in the maxillary molar region. The mucoperiosteal flap was reflected, and the original communication was visualized. The opening into the antrum was enlarged with rongeur forceps. The tooth was then delivered with an Allis clamp. The antrum was débrided, and the mucoperiosteal flap was closed with silk sutures.

Postoperative roentgenograms were taken (Figs. 15 and 16). The patient was continued on antibiotic therapy and sedation and was instructed regarding home care.

Healing was uneventful. Alternate sutures were removed one week postoperatively. Three days later the remaining sutures were removed.

One month following surgery healing was complete by primary intention, and the patient was referred to his family dentist.

Fig. 13.

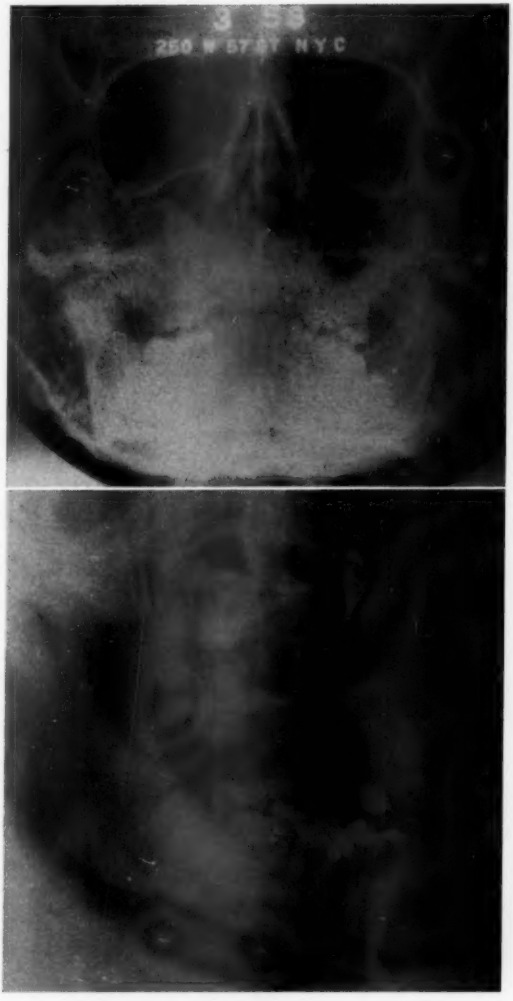


Fig. 14.

Fig. 13.—Case 1. Waters' projection revealing tooth in maxillary sinus.
Fig. 14.—Case 1. Profile roentgenogram revealing tooth in maxillary sinus.

Fig. 15.

Fig. 16.

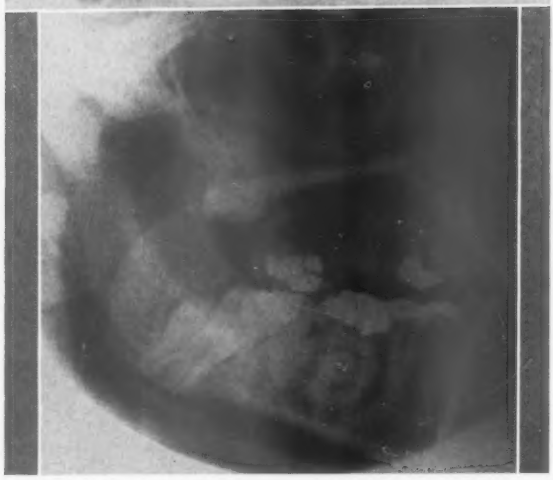


Fig. 15.—Case 1. Postoperative Waters' projection showing removal of tooth from maxillary sinus.

Fig. 16.—Case 1. Postoperative profile roentgenogram showing tooth removed from maxillary sinus.

CASE 2.—A 42-year-old white woman was referred by her dentist with a chief complaint of pain, inability to open her mouth, and swelling of one day's duration.

History of the present illness revealed that on the preceding day her dentist attempted to remove an impacted maxillary molar. During the procedure, the tooth was "lost" in the soft tissues of the face.

Fig. 17.

Fig. 18.

Fig. 17.—Case 2. Posteroanterior projection revealing tooth in infratemporal space.
Fig. 18.—Case 2. Profile roentgenogram revealing tooth in infratemporal space.

Clinical examination revealed extra-oral swelling over the left malar bone, extending across the zygomatic arch. The patient had a trismus and could open her mouth only about 1.5 cm. The soft tissue in the area of surgery was in a satisfactory state of repair.

Roentgenographic examination revealed that the third molar was displaced in the infratemporal space (Figs. 17 and 18).

The patient was placed on antibiotic therapy and sedation, and arrangements were made for hospitalization. On admission to the hospital, her medical history was essentially negative and noncontributory. Laboratory tests of the blood and urine were within normal limits.

On the following day, the patient was premedicated and taken to the operating room. Anesthesia was induced with Pentothal sodium and was maintained with nitrous oxide-oxygen and Pentothal sodium after nasotracheal intubation. The patient was prepared and draped in the usual manner. The mouth was suctioned and packed. An incision was made high in the mucobuccal fold, extending from the first molar to the tuberosity. By means of blunt dissection through the underlying tissue, the infratemporal space was reached medial to the coronoid process of the mandible. By gentle palpation, the tooth was identified and localized. It was then grasped with an Allis clamp and delivered. The incision was closed with interrupted silk sutures. The oral cavity was checked for hemostasis and suctioned, and the packing was removed. The patient reacted in the operating room and was returned to her room in good condition.

Fig. 19.—Case 2. Postoperative posteroanterior projection showing tooth removed from infratemporal space.

The patient was placed on antibiotic therapy and sedation, and recovery was uneventful. The postoperative trismus gradually diminished.

The patient was discharged from the hospital on the fourth postoperative day, to be followed as an outpatient.

One month following removal of the displaced third molar, opening and excursions of the mandible were normal and the patient was free of subjective or objective symptoms. A postoperative roentgenogram was taken (Fig. 19).

CASE 3.—A 46-year-old white woman was referred by her dentist for removal of an impacted maxillary third molar (Fig. 20). During an attempted removal two days prior to the patient's visit to this office, the tooth had been forced into the maxillary sinus by her dentist.

Clinical examination revealed an extraoral swelling over the left malar bone. The area was indurated and somewhat tender to palpation. There was some trismus. Intraoral examination revealed a buccal mucoperiosteal flap in the third molar region which was not sutured. The surgical site was tender to palpation, with ecchymosis and edema of the tissues.

Roentgenographic examination revealed the tooth in the maxillary sinus (Figs. 21 and 22).

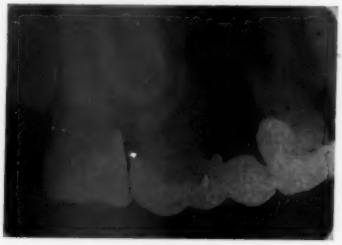


Fig. 20.—Case 3. Preoperative roentgenogram showing impacted maxillary third molar.

Fig. 21.

Fig. 22.

Fig. 21.—Case 3. Waters' projection showing tooth in maxillary sinus.
Fig. 22.—Case 3. Profile roentgenogram showing tooth in the maxillary sinus.

Because of the clinical symptoms present at the time of examination, removal of the tooth was postponed until these sympyoms disappeared. At the time of that first visit, however, the buccal mucoperiosteal flap in the third molar region was sutured under local anesthesia. The patient was placed on antibiotic therapy and sedation and was instructed to return in two days.

Her medical history was essentially negative and noncontributory.

When the patient returned, the swelling and trismus had diminished and she was relatively comfortable.

By the time of her next visit, the patient presented minimal swelling and trismus. The intraoral sutures were removed. An appointment was made for removal of the third molar from the maxillary sinus.

Fig. 23.—Case 3. Clinical photograph showing closure of the mucoperiosteal flap.

Fig. 24.—Case 3. Postoperative Waters' projection showing tooth removed from maxillary sinus.

Under local anesthesia consisting of an infraorbital and a posterior superior alveolar nerve block and infiltration, both buccally and palatally, a Caldwell-Luc incision was made from the canine to the second molar. The mucoperiosteal flap was reflected to expose the lateral wall of the maxillary sinus. A window was opened with surgical burrs and

was enlarged with rongeur forceps. The Schneiderian membrane was incised and reflected. The tooth was visualized and was removed with an Allis clamp. The mucoperiosteal flap was closed with interrupted silk sutures (Fig. 23).

A postoperative roentgenogram was taken (Fig. 24). The patient was continued on antibiotic therapy and sedation and was instructed with respect to home care.

Healing was uneventful, and the sutures were removed on the fifth postoperative day.

One month following surgery, the patient was free of objective or subjective symptoms and was referred to her family dentist.

SUMMARY

In the removal of impacted teeth, many problems may arise. The indications and contraindications for removal of the tooth at a particular time are best left to the judgment of the oral surgeon. Improper application of forces under poor surgical exposure may readily displace the tooth into the maxillary sinus, the soft tissues of the face, or the infratemporal space and may result in fracture or paresthesia.

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250 WEST 57TH ST.

ORAL MEDICINE

PROBLEMS OF ALLERGY ENCOUNTERED IN DENTISTRY

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INTRODUCTION

THE specific change in the reactivity of tissues to antigenic substances, known as allergy, is characterized by the production of specific antibodies. In man two types—hereditary and acquired—are generally recognized.

Among those with hereditary forms are patients with eczematoid or atopic dermatitis who often exhibit an inherent tendency to develop drug sensitivity; this often includes sensitivity to local anesthetics and penicillin.

The term *atopy* was introduced by Coca and Cooke to describe the hereditary sensitivities.[1] Later, Cooke and Vander Veer[2] showed that only the tendency to develop allergies was inherited. The specific reaction to an allergen is an acquired phenomenon and is dependent upon environmental exposures.

Anaphylaxis in experimental animals, an acquired form, has been the most extensively studied allergic phenomenon and serves as the basis for present concepts of allergy in man. It was shown that a guinea pig, for example, will not react to the first dose of allergen. However, a previously exposed and hence sensitized animal will react to a second injection of the same antigen. It was found that this state of sensitization could persist for two years.[1]

In man, anaphylaxis is an acquired sensitivity which differs from spontaneous reactions to allergens It is not a reaction influenced by familial tendencies, since sensitization always occurs with the first large antigenic dose. However, anaphylactically sensitive persons show characteristic skin and conjunctival reactions to very small amounts of antigen.[1] Although fatal reactions have followed minute test doses, careful testing with suspected agents remains the safest means for avoiding severe reactions.

Eczematoid or atopic dermatitis is an intrinsic form of allergic dermatitis. It is dependent upon the same hereditary factors that predispose to asthma, allergic rhinitis, and urticaria, and it is often associated with these conditions.[1]

From the Veterans Administration Hospital, West Roxbury, Massachusetts.

Patients with atopic dermatitis show immediate wheal reactions (10 to 20 minutes) when skin-tested with specific antigens. Introduction into the skin of small amounts of allergen by scratch or intracutaneous injection produces local vasodilation, increased capillary permeability, and transudation of fluid into the tissues. This is manifested by an itching wheal with surrounding erythema.

The case of a man with atopic dermatitis and known multiple allergies who was examined in the dental clinic and found to need important dental work is presented. It was felt that careful testing with suspected allergens must precede necessary dental treatment.

CASE REPORT

A 66-year-old retired carpenter was admitted to the West Roxbury Veterans Administration Hospital on Oct. 12, 1955, complaining of severe itching, chronic cough, and severe epigastric pain. His skin disease had been present almost continuously for the past seven years. In 1953 he had a gastrointestinal hemorrhage which was treated conservatively. An x-ray study at that time revealed a prepyloric ulcer. There was a history of episodes of pneumonia that responded well to penicillin. During his most recent pulmonary infection, the patient developed a skin rash with urticaria.

On physical examination, the patient appeared chronically ill. There were infection and edema of the left conjunctiva. His tongue was smooth, red, and shiny. The skin showed an extensive subsiding rash and chronic eczematoid dermatitis with scaling, edema, fissures, and hemorrhage (Fig. 1). The remainder of his skin was coarse and dry. The chest was emphysematous. There were diffuse coarse rhonchi at both bases. The abdomen showed slight right upper quadrant tenderness. There were signs of hypertrophic arthritis of the knees, hands, and ankles.

Laboratory and x-ray findings: The patient's blood and urine were normal on admission. X-ray studies revealed a diffuse increase in vascular markings throughout both lung fields and an incompletely obstructing indurated ulcer in the distal stomach.

Hospital course: The patient was admitted to the medical service and placed on an ulcer regimen. Routine dental examination revealed an edentulous maxilla with an ill-fitting upper denture and four carious lower teeth. Radiographs of the remaining lower teeth showed extensive caries with periapical disease. Extraction of the remaining lower teeth and construction of full upper and lower dentures were recommended. The patient recalled vomiting and mentioned other gastrointestinal symptoms following a previous injection of Novocain for extraction of one tooth. He was skin-tested intradermally with 2% procaine and with Xylocaine; in both instances an itching wheal appeared within ten minutes. A few days later Unicaine was injected intradermally without reaction. On Jan. 24, 1956, his remaining lower teeth were extracted with Unicaine. The patient was transferred to the surgical service on March 28, 1956, and a subtotal gastrectomy was performed five days later. He was discharged on April 23, 1956, to be followed in the gastrointestinal clinic.

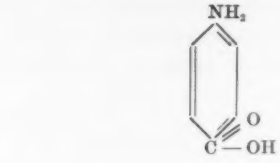
DISCUSSION

In considering specific sensitivities resulting from local anesthetic agents, it appears that the allergic potentials of these drugs vary according to differences in their chemical structures For example, p-aminobenzoic acid derivatives seem to be the most antigenic.[3, 6] Procaine, Monocaine, Pontocaine, Larocaine, and Tutocaine fall into this group (Fig. 2). The ortho-substituted

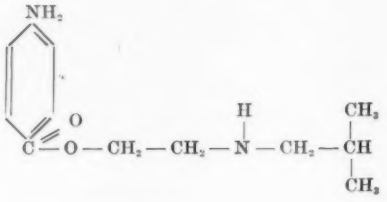
A.

B.

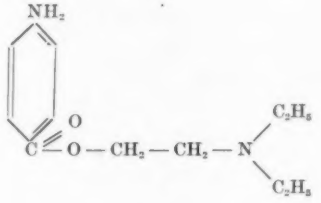
Fig. 1.—*A*, Skin of hands. *B*, Skin of body. These illustrations show eczematoid dermatitis with scaling, edema, fissures, and hemorrhage.



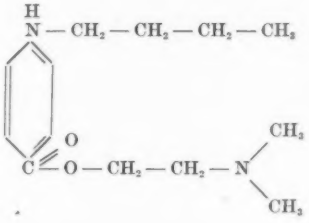
p-aminobenzoic acid



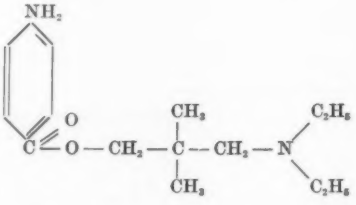
Monocaine



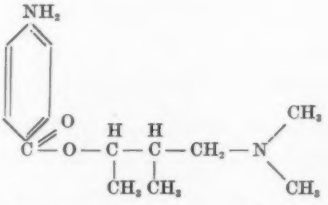
Procaine



Pontocaine



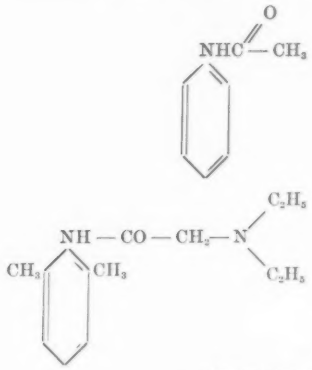
Larocaine



Tutocaine

Fig. 2.—Chemical structures of local anesthetics derived from p-aminobenzoic acid.

compounds appear the least antigenic, and the meta-substituted aminobenzoic acid derivatives are intermediate.[7] Xylocaine, or lidocaine, is an ortho derivative of acetanilid (Fig. 3). The increased antigenic effect of compounds in



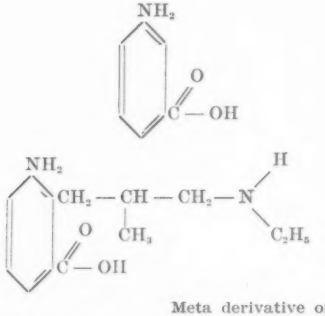
Acetanilid

Xylocaine
or
Lidocaine

Ortho derivative of acetanilid

Fig. 3.—Chemical structure of local anesthetic derived from ortho-acetanilid.

which the para-hydrogen atom is replaced by a larger atom or group has been explained on the basis of increased Van der Wall's interaction between the substituent and the antibody.[7] Unicaine is a meta-aminobenzoate (Fig. 4).

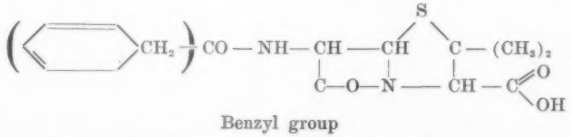


Meta-aminobenzoic acid

Unicaine

Meta derivative of benzoic acid

Fig. 4.—Chemical structure of local anesthetic derived from meta-aminobenzoic acid.



Benzyl group

Fig. 5.—Chemical structure of penicillin G or benzyl penicillin.

Goodman[8] found that there was no reaction to drugs of this structural type in which the amino group was in either the ortho or the meta position with respect to the carboxyl group.

Although penicillin has antigenic properties, the actual mechanism of sensitization in man is not clearly understood. Penicillin G or benzyl penicillin has little in its molecular structure to help explain the allergic reactions[9] (Fig. 5). The underlying mechanism might resemble that of other atopic allergies as, for example, atopic dermatitis.[9]

SUMMARY

In cases of atopic dermatitis the inherent tendency to develop allergies must be recognized. The history of the case reported here revealed sensitivity to Novocain and penicillin, which was verified with skin tests. Additional skin tests showed an allergic response to Xylocaine but no reaction to Unicaine. The difference in the chemical structure of these local anesthetics is discussed.

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ORAL PATHOLOGY

INVAGINATED TEETH (DENS IN DENTE): CONTENTS OF THE INVAGINATION

Martin A. Rushton, M.D., Odont. D., F.D.S., London, England

TEETH showing abnormal developmental invaginations have been given many names, including dens in dente, dilated odontome, gestant odontome, etc. An excellent historical review of the literature is given by Gustafson and Sundberg,[2] commencing with the first detailed description by John Tomes[8] 100 years ago. Gustafson and Sundberg note that Moral[4] concluded that the internal enamel-lined cavity must have been filled originally by part of the enamel organ and that this must have received its nutrition from somewhere, presumably through the orifice of the invagination on the surface of the crown of the tooth. Some have also believed that part of the blood supply might come from the pulp through a communication between the invagination and pulp cavities.[3] In cases in which the invagination passes right through the tooth to the periodontal membrane, it is clearly possible that a source of supply may be acquired from the latter also.

In nearly all of the very numerous specimens described, the tissue within the invagination cavity has either been necrotic at the time of examination or has disappeared entirely. There are several reasons for this. The earlier specimens were examined dry some time after their removal, usually by making ground sections. Specimens were not commonly removed from the patient until an alveolar abscess had occurred. Even when they were removed after eruption, for cosmetic or other reasons unconnected with infection, the tissues within the invagination cavity were still found to be necrosed although sometimes only recently dead, so that it was possible still to recognize fibrous tissue as well as bonelike substances and, of course, enamel. This early death of the soft tissues within the invagination has been reasonably explained in two ways. When the tooth erupts, the invagination orifice, which is usually on the surface of the crown, can no longer provide a channel through which a blood supply can enter. In addition, the exposure of the orifice to mouth fluids allows infection to enter the invagination cavity.

In one of Gustafson and Sundberg's cases (No. 6), described previously by Bøhn,[1] the tissues were evidently partly vital. According to Bøhn, this upper lateral incisor from an 18-year-old girl had been erupted for ten years, had a granuloma on its partly resorbed root, and the pulp contained granulation tissue and showed reticular atrophy and "heavy necrotic degeneration." In the invagination cavity there were foreign bodies, such as cellulose fibers, abundant connective tissue, and necrotic enamel pulp with cholesterin crystals. According to Gustafson and Sundberg, however, the contents of the cavity were not necrotic but were debris of the enamel organ with the ameloblasts well preserved, and there were channels containing blood vessels passing from the pulp to the invagination cavity. Presumably this specimen had undergone chronic inflammatory changes for many years. Oehlers[5] has reported a similar case.

Descriptions of specimens removed before eruption, in which the soft tissues were still alive and in which changes such as inflammation and resorption had not yet taken place, are not known to me. For this reason and because several authors, including myself,[6] have in the past published speculations on what they thought should be found, three specimens will be described. They were all derived from the anterior part of maxillae and show various stages of development, though unfortunately not the earliest stage which we should most like to see. Two were somewhat damaged at operation.

Specimen 1.—This was a supernumerary tooth obstructing the eruption of the left upper central incisor in a child of 7½ years. It was a partly formed conical tooth with an invagination channel passing vertically through it from a small spot near the tip of the cone. No root had yet formed and the calcified part formed a ring around the invagination. When the tooth was decalcified and sectioned, this showed as a mass of dentine on each side of the central invagination, each mass shaped rather like an arrowhead (Fig. 1, *D*, *D*) and originally covered with enamel. Between these a thin strand of soft tissue (*A*) containing blood vessels and epithelium runs vertically and expands somewhat at its deeper portion. The deepest part of the invaginating tissue must have been in contact with the dental pulp (*P*), but the area of contact is not shown, owing to damage at operation. It can be seen, however, that there is an epithelial diaphragm, composed of external enamel epithelium and stellate reticulum, separating the invagination from the pulp (Figs. 1 and 2). The later-formed enamel at the deepest part of the invagination and the neck of the tooth was very immature (*E*), more so within than without as is usually the case. At the deepest part of the invagination, where the formation of enamel matrix was still continuing, the various layers of the enamel organ can be distinguished (Fig. 2), but the center of this part is empty and if it ever contained anything but fluid this has been lost. In the more superficial part the soft tissue is a thin cord covered externally with columnar or cuboidal enamel epithelium and composed of other epithelium supported by a very scanty connective tissue containing numerous thin-walled vessels but practically

no fibrous tissue (Fig. 3). It contained also irregular bodies, to some of which ameloblasts are applied, which represent discrete deposits of enamel matrix.

This, then, is a specimen in which the formation of enamel and, of course, dentine had not yet ended and in which the invagination contained enamel organ of various degrees of maturity, a blood supply passing through the invagination channel, and practically no fibrous tissue.

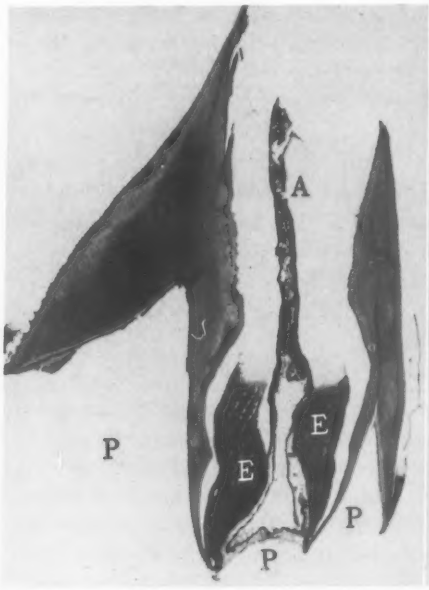

Fig. 1.

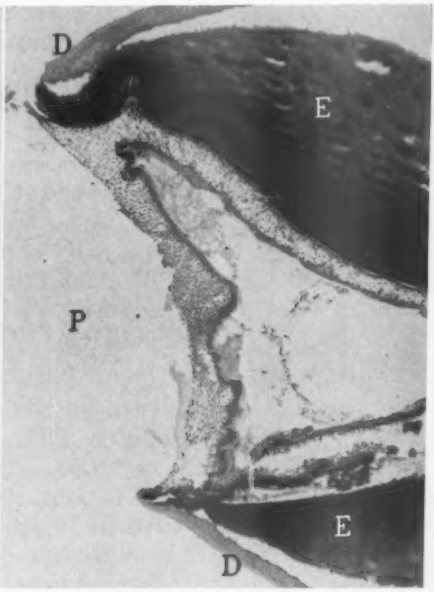

Fig. 2.

Fig. 1.—Specimen 1. *A*, Cord of vessels and epithelium occupying the invagination channel; *D*, dentine; *E*, immature enamel; *P*, space occupied by pulp which was torn away during the removal of the tooth. (Hematoxylin and eosin stain. Magnification, ×12; reduced ⅛.)

Fig. 2.—Deepest part of the invagination shown in Fig. 1. Ameloblasts are in contact with the enamel matrix, and the stellate reticulum and external enamel epithelium can be seen forming a sort of diaphragm. The tissues are a little disturbed at the lower right, and it is impossible to say whether anything other than fluid occupied the central space. (Magnification, ×50; reduced ⅛.)

Specimen 2.—This specimen had reached a slightly more advanced stage than the last, in that the deepest part of the invagination cavity had recently become closed by a layer of dentine, shutting it off from the pulp (Fig. 4). The tooth was a supernumerary structure and had been removed from a girl of 8½ years because it was preventing the eruption of the upper incisors. In the invagination cavity the formation of enamel matrix had ceased and, except at the deepest part, maturation was well advanced. The invaginating tissue is seen as a rather swollen cord covered with columnar enamel epithelium and containing other epithelium and rather large thin-walled blood vessels (Fig. 5).

Fibrous connective tissue elements are very scanty, and the striking feature is that most of the space was occupied by fluid in which strands of epithelium appeared to float (Fig. 5). The soft tissue is constricted where it passed through the orifice of the invagination cavity.

Specimen 3.—The third specimen had reached almost full development but was not yet erupted. It was removed from the upper lateral incisor region. The whole structure was pear-shaped, with the invagination entering through a channel in the conical enamel-covered crown. The invagination cavity was rather large (about 10 mm. by 7 mm. by 7 mm.) and was chiefly filled with fluid, although it also contained living tissue (Fig. 6). The pulp was healthy.

Fig. 3.

Fig. 4.

Fig. 3.—Contents of the invagination channel near *A* in Fig. 1 consist of a tube of epithelium with ameloblasts on its surface and blood vessels within but little connective tissue. (Magnification, ×110; reduced ⅛.)

Fig. 4.—Specimen 2. The entrance to the invagination cavity is at top right, and the cavity contains living tissue. Mature enamel has been removed in preparation, but immature enamel matrix has remained in the deepest part of the cavity which is separated from the pulp by a thin wall of dentine. Part of the pulp and dentine shown on the left of the figure was torn away at operation. (Hematoxylin and eosin stain. Magnification, ×8; reduced ⅛.)

The entrance to the invagination was rather tortuous, so that it cannot be followed entirely in any one section; it was occupied by a thin cord of tissue covered with ameloblasts and contained epithelium, enamel droplets, and rather large thin-walled vessels. There was very little connective tissue. The main invagination cavity was partly lined with enamel, of which organic elements remain in the section, and much of this is covered with a cuboidal or

flattened layer of ameloblasts. In other places the ameloblasts have disappeared. The main part of the cavity is occupied by fluid forming a cystlike collection (Fig. 6, *B*) bounded partly by the walls of the cavity with their epithelial covering and partly by a compressed tenuous fibrous tissue (Fig. 7). Into this fluid there bulges a nodule of soft tissue in which there have been epithelial proliferation and leukocytic infiltration (Fig. 8). The dentine separating the invagination cavity from the pulp has imperfections, including small channels, but these did not include recognizable blood vessels.

Fig. 5.

Fig. 6.

Fig. 5.—Enlarged view of the soft tissues within the invagination cavity. There is a surface layer of ameloblasts which was in contact with the enamel, other dental epithelium, and rather large thin-walled blood vessels. The tissues were distended with fluid in which strands of epithelium appeared to float. (Magnification, ×50; reduced ⅛.)

Fig. 6.—Specimen 3. The entrance to the invagination cavity containing a vascular epithelial cord is in the region of *A* but is not seen in this section. The bulk of this cavity was filled with fluid of which the protein content has shrunk in preparation (*B*). To the right of *B* and below it are vital soft tissues shown better in Figs. 7 and 8. *P*, Healthy pulp. (Hematoxylin and eosin stain. Magnification, ×6; reduced ⅛.)

These few specimens show what was, indeed, to be expected: that the invaginating tissue consisted of epithelium from the enamel organ with a generous vascular supply from the follicle. There was little supporting connective tissue in these cases, but it is well known from necrotic specimens that sometimes there is much more, including bony masses. An interesting feature is the accumulation of fluid in the invaginating tissue, an event which evidently can start quite early. The significance of this will be discussed.

Mr. Ivor Kramer has very kindly allowed me to examine his collection of sections of invaginated teeth, including three specimens in which the tissues

in the invagination cavities were still vital and not infected. The appearances in these latter specimens resembled those described here in specimens 2 and 3, and in all of them there were collections of fluid lying in the connective tissue and occupying a large part of the invagination cavity. Intermediate stages between edematous connective tissue and such collections could be seen.

Fig. 7.

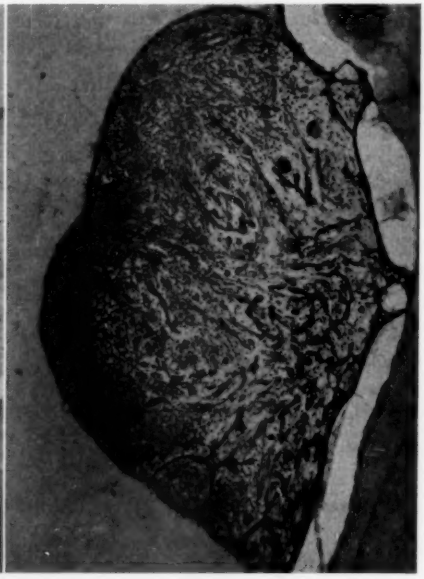

Fig. 8.

Fig. 7.—Floor of the fluid collection bounded by compressed delicate connective tissue (below *B* in Fig. 6). Beneath this is dentine overlying the pulp and showing scraps of enamel matrix on its surface and small channels in its substance. (Magnification, ×70; reduced ⅛.)

Fig. 8.—Nodule of soft tissue projecting into the fluid collection near *B* in Fig. 6 and lying on the surface of the enamel lining. It shows epithelial proliferation and infiltration with leukocytes. (Magnification, ×45; reduced ⅛.)

DISCUSSION

Gustafson and Sundberg refer to several tenable hypotheses about the causation of these deformed teeth—those of "active invagination," "retardation," and their own hypothesis. They make a distinction between invaginated teeth which they call "dens in dente" and those which they call "odontomata," but they add: "The assumption, made by some authors that deep foramina caecum [sic], dens in dente, and odontomata only show quantitative differences, appears correct."

If we consider the hypothesis of retardation first, Kronfeld[3] wrote: "Orban has shown that the growth of tooth germs occurs on the whole in a centrifugal direction. This suggests that a dens in dente may develop not by actual *ingrowth* of enamel organ into underlying pulp, *but by relative retardation in growth of a portion of the enamel organ*, possibly at or near the lingual

pit, while the surrounding dental tissues continue to expand and grow in a peripheral direction." A great objection to this as an explanation of any but the smaller specimens is that many invaginated teeth are larger than the expected size of the normal teeth which they replace. Many, indeed, are so obviously enlarged by some central expansion that they have long been known as dilated odontomes. No amount of retardation of growth of part of an enamel organ could by itself produce an enlarged tooth. Again, if the invagination were due to retarded growth alone it would be unexpected that the diameter of the earliest formed part of the invagination cavity—a ring of dentine surrounding the invagination channel at the surface of the tooth—should be almost invariably much less than that of the later formed deeper parts of the cavity. There is the further important objection of Swanson and McCarthy[7] regarding the depth of the enamel in the invagination which often extends rootward of the cervix of the tooth.

These objections do not rule out local retardation as a factor in the initiation of the anomaly if the expansion of the invagination could be attributed to another factor. But the retardation might not be in the growth of the enamel organ so much as in that of part of the dental papilla, upon the expanding surface of which the enamel organ should be carried centrifugally and upon which the inner enamel epithelium is said to rely for nutrition until dentine formation begins.[9]

Gustafson and Sundberg consider that the theory of retardation is correct and, as a complement to this theory, they suggest that localized absence of ameloblasts is the primary cause of the malformation. "If no ameloblasts develop, there can be no dentine. In that way there is a direct communication right through the dental bud and, as the connective tissues lie as a core in the defect, the defective part is held back in its original place." There is rather little evidence for this interesting conception, and the same objections apply as in the case of Kronfeld's hypothesis. Moreover, a failure of dentine formation need not be due to absence of ameloblasts; it could also be due to a functional defect of the enamel epithelium or to an inability of the dental papilla to respond. Any gap in the dentine separating the invagination cavity from the pulp is at the last-formed and deepest part of the invagination cavity; it is in relation to tissues which by that time may well have impaired or altered functional capacity.

The specimens recorded here show that at the time of examination the connective tissue core consisted of almost nothing but thin-walled, unsupported blood vessels although this may not always be the case. And the first specimen suggests, although it does not prove, the presence of a complete diaphragm of epithelium between the invagination cavity and the pulp at a rather early stage.

With regard to the hypothesis of active invagination of the enamel epithelium into the dental papilla, one which I have previously supported,[6] I now feel that there is no evidence that this is an initial phase of the malformation, although it might be, but there is strong evidence that when the invagination

has begun it has often become the site of a vigorous expansion which has distorted the form of the tooth to varying and sometimes extreme degrees. It is quite possible that the mechanism which could produce the initial invagination and that which produces the expansion are quite different. Bøhn made the suggestion that the expanding force was increased tissue pressure caused by stasis. The cause of the stasis would be the compression of the soft tissues in the entrance channel of the invagination by the formation of enamel in a circle around it. Oehlers[5] also seems to favor this view.

Fig. 9.—Invagination channel of an invaginated tooth. The width of the channel between the dentine walls (*1*) is about 1.1 mm. The width between the enamel surfaces (*2*) is about 0.1 mm. (Hematoxylin and eosin stain. Magnification, ×50; reduced ⅛.)

The material presented here is in accordance with that conception in that the accumulation of fluid in the invaginating tissues has been demonstrated. The narrowing of the invagination channel entrance (what Bøhn calls the "bottle neck") by the concentric apposition of enamel is well shown in many specimens (for example, Fig. 9, in which it can be calculated that the width of the channel was at one time 1.1 mm., the measurement between the internal dentine walls). On each side about 0.5 mm. of enamel was deposited, leaving a final channel of not more than 0.1 mm. at its narrowest part, less than one-tenth of the original diameter. This process could well lead to venous congestion and sometimes transudation of fluid into the invagination cavity due to raised venous pressure and increased permeability of the vessel walls. Also, it could possibly lead to increased growth of tissues within the invagination.

This venous congestion and transudation could occur sufficiently early to affect the shape and size of the invagination cavity, for the narrowing of the channel entrance can be complete at quite an early stage and certainly before dentine has outlined much of the invagination cavity (as in specimen 1). Venous congestion, of course, could not begin until *inter alia* blood vessels had entered the invagination from the follicle, for at an earlier stage the invagination would contain only enamel organ, chiefly stellate reticulum. We are told, however, that the thickness of the stellate reticulum is greatly reduced when the first layers of dentine are laid down (Orban, 1953), so that it is at the time when a ring of calcified tissue is beginning to be deposited around the neck of the invagination that the blood vessels may be expected to be entering it.

The danger of starting this suggested mechanism of internal expansion by venous congestion would presumably arise in a tooth germ in places where there was a deep narrow entry of the enamel organ into the dental papilla (whether as a result of some interference with growth or as a result of slight variations of the normal pattern) at the time that dentine formation begins at the same part.

It is probably essential to this hydrostatic hypothesis that the sole blood circulation should enter and leave the invagination by the "bottle neck" during the formation of the tooth, for if there were a free alternative circulation through the dental pulp at this stage there would be no reason for venous congestion to occur. In the early specimens described here, no vascular communications between the pulp and the invagination cavity were found, although in the absence of complete serial sections they cannot be excluded. More complete examination of early specimens should make it clear whether the vascular communication seen by Gustafson and Sundberg and others in infected specimens is found also at an early stage or whether the vascular territories of the pulp and the invagination are then separated by enamel organ.

SUMMARY AND CONCLUSIONS

1. Three specimens of unerupted and uninfected invaginated teeth, in which the invagination contained living tissue at the time of removal, are described. Only one of these had completed its development.

2. The soft tissue contained in the invaginations consisted of enamel epithelium with a core of blood vessels but little other connective tissue.

3. No communication between the blood vessels of the pulps and those of the invagination was observed, although this cannot be excluded.

4. Two specimens showed accumulations of extravascular fluid within the invagination, and the same was seen in the three uninfected specimens belonging to Mr. I. Kramer.

5. The mechanism of the malformation is discussed. It is contended that while there is still insufficient evidence for a conclusion as to how the process

of invagination is initiated, when once started it could be rendered irreversible and extended by a hydrostatic mechanism as suggested by Bøhn.

I am much indebted to Mr. J. E. Hutchinson for the histologic preparations and photomicrographs, and to Mr. J. F. Dudley for other sections; also to Mr. Ivor Kramer of the Postgraduate Dental Institute, University of London, for the loan of specimens.

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THE RELATIONSHIP OF GLOBULOMAXILLARY CYSTS TO THE FUSION OF EMBRYONAL PROCESSES AND TO CLEFT PALATES

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INTRODUCTION

GLOBULOMAXILLARY cysts are known primarily from the studies of Roper-Hall[12, 13] and Thoma.[17] These extradental cysts occur very rarely. Akira and Kitamura[1] succeeded in finding in the literature of 1937 to 1952 but twelve reports of such cases. Practically all these cysts develop in the region of the teeth and are seen on roentgenograms to project over the teeth, seeming to be cysts attached to them. An incorrect diagnosis may thus be followed by the superfluous devitalization or extraction of the teeth.

Cysts of nondental origin comprise, according to Thoma, the following two groups: (1) nasopalatine cysts, which develop in the incisive canal from epithelial remnants in the duct, and (2) fissural cysts having two forms—median and globulomaxillary.

It was Klestadt[7] who suggested that epithelial cells remaining at the site of fissures between the embryonic processes lead to cyst formation at a later time; hence, the term "fissural cyst" was applied to them. If these embryonic processes fail to fuse, facial and maxillary clefts corresponding to these sites may form; hence, the term "facial cleft cyst."

The median cysts develop in the midline at a site corresponding to the point of fusion of the two globular processes. Here if the fusion fails to take place, which is rare, the median cleft palate also forms.

The globulomaxillary cysts, on the other hand, form laterally from the midline, between the canine and the lateral incisor. According to most of the reports in the literature, they form at the junction of the globular and maxillary processes where the premaxilla joins the maxilla. At this same place the alveolar clefts occur; thus the site of globulomaxillary cysts and of the cleft palate is the very place where the premaxilla joins the maxilla.

Whereas the median cysts really develop at the point of fusion of the globular processes of the two sides and at the site where the median cleft of the palate occurs, the statements regarding the relationship of globulomaxillary cysts to the fusion and the clefts and the relationship of clefts to the

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junction of the premaxilla and the maxilla are not quite in agreement with our embryologic knowledge and roentgenologic observations. I wish to call further attention to this phenomenon.

CLINICAL AND EMBRYOLOGIC STUDY OF THE CLEFTS

The first task is to study the embryologic aspect of the relationship of clefts to the teeth and to the bony substance, that is, the relationship of clefts to the fusion of the premaxilla and the maxilla.

There has been a very long discussion on the relationship of clefts (that is, the fusion) to the teeth and to the bony substance, respectively. The view was held for a time that this corresponds to the point where the premaxilla joins the maxilla and that the cleft forms as a result of failure of ossification.[8] This view would be correct only if the premaxilla forming the entire maxilla developed from the globular processes.

The maxilla and the mandible are formed in different ways. The mandible develops from two embryonal processes (the mandibular processes) whereas the maxilla is formed from two pairs of embryonal processes (namely, the globular processes, which make up the protruding parts of the medial nasal process, and the maxillary processes). Thus, two separate bones are formed at ossification—the premaxilla (or os incisivum) and the maxilla—and after ossification of the two the complete maxilla is formed. The point of junction of the premaxilla and the maxilla (that is, the incisive suture) can be well seen on the skull of the newborn infant between the lateral incisor and the canine tooth; at a later period it disappears, however, and the two bones fuse perfectly. With some animal species in which the premaxilla had developed very definitely, the incisive suture is retained during later life as well.

Inouye,[5] Jarmer,[6] and Peter[10] have stated that the premaxilla is formed not only from the globular process, but that the maxillary process also takes part in its formation. Thus, the point of fusion of the globular and maxillary processes and the site of clefts, respectively, cannot correspond to the junction of the premaxilla and maxilla where the incisive suture is found. This latter is located between the lateral incisor and the canine tooth, and the fusion and the clefts, respectively, are to be found more medially in the direction of the lateral incisor (Fig. 1).

Since the fusion and the clefts, respectively, develop at an earlier date than the ossification, depending on the development of the bony parts, the site of the clefts varies if related to the location of the teeth. Kölliker[8] examined fifty-four cases of cleft palate and found the cleft to occur between the lateral incisor and the canine tooth in twenty-six cases, between the central and lateral incisors in twenty-three cases, and between the central incisor and the canine tooth in five cases when the lateral incisor was absent.

Different anomalies of the lateral incisors (for example, their absence or rudimentary or supernumerary formation) accompany clefts in a great many cases. Millhon and Stafne[9] observed that in about eighty-two cases with cleft palate a supernumerary lateral incisor occurred in twenty-two cases and this tooth was absent in seven.

It is well known that the primordia of the teeth are already formed before the fusion takes place. The primordium of the lateral incisor is affected if the fusion fails to take place, since it may become divided, as at an early stage (in the reversible phase of determination) a much higher developmental potency exists and each half is able to produce a tooth. If this dichotomy of the tooth occurs later, at the irreversible stage of determination, both the two half-germs may degenerate and no lateral incisor will develop according to Brandt.[2]

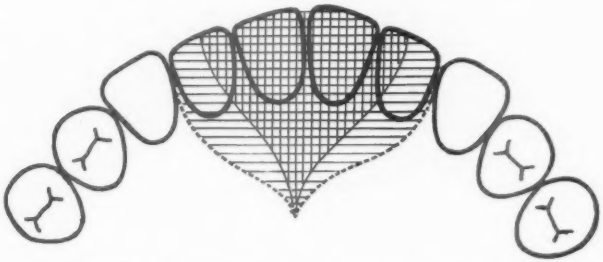

Fig. 1.—Development of the premaxilla. It extends between the lateral incisor and the canine to the incisive suture. The checkered part forms from the globular process, the cross-lined portion from the maxillary process. After a figure in Fischel: *Entwicklung des Menschen*, Wien and Berlin, 1929, Julius Springer.

ROENTGENOLOGIC EXAMINATION OF THE CLEFTS

Roentgenograms showing the relationship between clefts and teeth agree with the aforementioned embryologic statements. Thus, the cleft may be located between the central incisor and the lateral incisor (Fig. 2), between the lateral incisor and the canine (Fig. 3), or between the central incisor and the canine (Fig. 4). Besides the clefts, anomalies of the lateral incisor may also be discovered by roentgenologic examination. The tooth may be absent, or it may show a rudimentary development. There is a third possibility, namely, that only the deciduous teeth erupt and often a supernumerary lateral incisor is encountered. In such cases the cleft is to be found between these two teeth (Fig. 5).

ROENTGENOLOGIC EXAMINATION OF THE GLOBULOMAXILLARY CYSTS

On the basis of roentgenograms, it can be stated that the typical location of the globulomaxillary cysts is between the lateral incisor and the canine, at the site that corresponds to the incisive suture. These cysts can be easily diagnosed in the developmental stage, but when they have already overgrown the apex of the adjacent root they cause divergence of the adjacent teeth and have a characteristic pear-shaped projection (Fig. 6). Such an appearance is a good sign to differentiate them from radicular cysts, especially if vital adjoining teeth exist.

Distinguishing small and quite large cysts, however, is a more difficult task. The small cysts are similar to the lateral periodontal cysts; hence, in this stage of development they can be diagnosed only beside vital teeth.

Fig. 2.

Fig. 3.

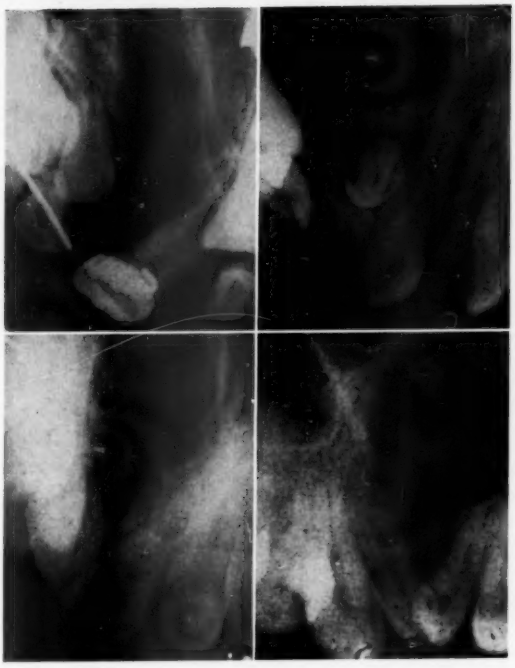

Fig. 4.

Fig. 5.

Fig. 2.—Cleft between the central and lateral incisors.
Fig. 3.—Cleft between the lateral incisor and the canine.
Fig. 4.—Cleft between the central incisor and the canine.
Fig. 5.—Cleft between the lateral incisor and a supernumerary lateral incisor.

Fig. 6.—Globulomaxillary cyst between the lateral incisor and the canine.

When the cysts have grown in every direction and have encroached upon the adjoining teeth as well, their roentgenologic appearance is very similar to that of a radicular cyst. In such cases globulomaxillary cysts can be diagnosed only if the adjacent teeth are vital. If the lateral incisor is absent, the roentgenologic appearance resembles a residual radicular cyst.

The cleft palate may have an irregular projection on the roentgenogram, or one similar to a cyst. If the cleft is between the lateral incisor and the canine, its appearance often resembles a globulomaxillary cyst, and if it is nearer to the median line its picture is rather like that of a radicular cyst. The diagnosis by clinical examination of the cleft is not difficult and, therefore, a false diagnosis is made only if it is based on the roentgenogram alone.

On the other hand, concealed clefts covered by soft tissues may occur. These can be diagnosed only on the basis of roentgenograms. Such concealed clefts associated with harelip were reported by Broderick.[3] I could not find reports of such cases by other authors in the literature.

SUMMARY

The statement that gobulomaxillary cysts form at the fusion of the globular and maxillary processes and at the place of facial clefts, respectively, disregards embryologic and roentgenologic findings. The site of the clefts and of the fusion, respectively, is not at the junction of the premaxilla and maxilla, but varies between the central incisor and the canine, being most often in the location of the lateral incisor. The globulomaxillary cysts, on the other hand, form in fact typically at the junction of the premaxilla and maxilla, that is, between the lateral incisor and the canine.

From all these findings, conclusions may be drawn with respect to the etiology and terminology of these cysts. The term "facial cleft cyst" appears to be illogical since the cyst does not form at the site of the clefts; the same is true for the term "globulomaxillary cyst," since this cyst does not form at the junction of globular and maxillary processes. The term "premaxilla-maxillary cyst" would be more exact in expressing the location.

On the basis of the foregoing facts, an etiological relationship between the cysts and clefts is rather improbable, although several authors allege that such a relationship exists. Thus, Scott[14] carried out examinations in fetuses and demonstrated microscopic cysts which, in his opinion, can cause clefts. The very rare cases in which a globulomaxillary cyst formed on one side and a cleft formed on the other refer to some relationship of this kind.

Assuming that the fusion is not where these cysts develop, the question may arise as to whether they originate from embryonic epithelial remnants at all. Robinson[11] holds the view that embryonic epithelial remnants cannot exist in the sutures and that the cysts originate in the primordium of supernumerary teeth.

To clarify the etiology of globulomaxillary cysts, further investigations are required. The problem, however, of their relationship to the fusion of embryonic processes and clefts, respectively, may be considered solved.

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ENDODONTICS

EFFECTIVENESS OF A GROUP OF ENDODONTIC THERAPEUTIC AGENTS

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INTRODUCTION

THE expanded development of clinical endodontics is dependent, in large measure, upon a re-evaluation of existing methods and treatment adjuncts routinely employed and their relationship to newer concepts.

This study represents a series of in vitro experiments designed to determine the effectiveness of certain root canal medicaments. These selected agents have been tested singly and in combination with some specific water-in-oil emulsifying bases.

The term "sterile canal" has been used widely, if not loosely, and is generally accepted as one of the critical and desirable conditions to be produced prior to the obturation of the pulp cavity with suitable filling medium. In the laboratory, however, it appears that the principle observed and recorded is the effectiveness of the "sterilizing agent" (medicament) against the growth of microorganisms.

The effectiveness of an agent or combination of agents and/or procedures (namely, electrosterilization, root resection, drug therapy, etc.) does not necessarily seem to produce a "sterile pulp cavity"; rather, these agents or methods act against bacterial elements and their products. Similarly, by acting against microorganisms and their toxic products, tissues which have been invaded or destroyed by the above are relieved of "toxic pressure." The reparative mechanism of the body is thereby permitted to function freely in the process of healing and resolution of existing periapical lesions.

By such action against infective agents and their sequellae within and beyond the pulp cavity, the "inner structure" of the tooth is rendered "bacteria free" and thus a passive or neutral condition is established within the pulp cavity.

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This action has been considered the production of a "sterile canal." By our present methods of bacteriologic control, this has been accepted as necessary prior to obturation of the pulp cavity.

The present study is a comparative in vitro experiment to record the effectiveness of agents routinely used in clinical endodontics. Water-in-oil suspensions as vehicular bases for some of these medicaments have been included in the study. It has been shown that the water-in-oil suspension seems to enhance the stability of certain of the drugs tested.

In combination with "ointment bases," some of the drugs tested were released slowly rather than rapidly. In ointment form, "drug potency" was allowed to remain at a high degree of efficient action. Dilution of the therapeutic agent is also prevented.

REVIEW OF LITERATURE

The dental literature reveals in vitro experiments conducted by Bartels.[1, 2] These studies emphasized that "formocresol, beechwood creosote, oil of cinnamon, eugenol and azochloramide do inhibit bacterial growth." Penicillin was found to be "affected" by zinc oxide and eugenol when used against *Staphylococcus aureus.*

Buchbinder and Bartels[3, 4] collaborated in a study evaluating antibiotic therapy. This work criticized the use of root canal cultures with antibiotic treatment. The critique was based largely upon the inaccuracy of bacteriologic culture methods in relation to antibiotics.

The contributions of Grossman,[5] Stewart,[6] Bender and Seltzer,[7, 8] and others have immeasurably increased interest in endodontic therapy.

The literature contains considerable material, both experimental and clinical which has sought to evaluate the effectiveness of specific drugs or combinations of drugs and antibiotics in the management of the pulp cavity.[9-17]

A portion of this study refers to the use of hydrophilic absorption ointment bases in combination with commonly used therapeutic agents. Some reference to the dermatologic literature is herewith included.

Hydrophilic ointments are substances containing complex high molecular hydroxyl animal fats. They are used as emollients or skin detergents in superficial skin infections and applied topically in pure form or as ointment bases in combination with other medicaments.

Perrin and Halpern[18] reported a comparative study of these bases in combination with commonly used dermatologic agents. Pertinent conclusions of this study revealed: "The water-in-oil emulsion is a desirable dosage form for topical therapy that may be used with all medicaments."

"This study demonstrates . . . the general desirability of the water-in-oil emulsion. Perhaps the reason for this lies in the *mechanism* of *drug release from the base,* whereby the active ingredient must traverse the continuous oil phase before it can be liberated to the treatment area. The *thickness* of this oil film would then be a *determinant* in the *drug release* and therefore would tend

to regulate the concentration of the drug diffusing into the area. In this manner a slower, more steady drug release results and there is a *minimum diffusion* of a large drug concentration which might be *irritant* to the skin."

MATERIALS AND METHODS

The in vitro study consisted of the preparation of a selection of medicaments, antibiotics, and polyantibiotic combinations, singly and in combination with water-in-oil emulsifying bases. These are listed in Table I. Sterile disks were impregnated with the various therapeutic agents as listed. Nonimpregnated sterile disks were used as controls.

TABLE I

THERAPEUTIC AGENT	RADIUS OF ZONE OF INHIBITION (MM.)	
	24 HOURS	72 HOURS
Control: No disk	—	—
Sterile disk	2 S	2 SD
1. Oil of cloves with 25% chlorobutanol without ointment base	2	2
2. Camphorated paramonochlorophenol without ointment base	5	6
3. Formocresol without ointment base	15	15 D
4. Cresolated formaldehyde without ointment base	13	13
5. Beechwood creosote without ointment base	4	6 S
6. Oil of cloves with 25% chlorobutanol with anhydrous lanolin	1	5 S
7. Oil of cloves with 25 per cent chlorobutanol with Aquaphor	4	4 S
8. Camphorated paramonochlorophenol with anhydrous lanolin	4	4 S
9. Camphorated paramonochlorophenol with Xylocaine ointment	4 D	4 SD
10. Camphorated paramonochlorophenol with Aquaphor	2 S	4 S
11. Formocresol with Xylocaine ointment	4	4 S
12. Formocresol with Aquaphor	12	14 D
13. Beechwood creosote with Aquaphor	—	4 S
14. Terramycin-cortisone ointment	1.5	6 SD
15. Tetracyline with Aquaphor	6	6 D
16. Grossman's PBSC formula	10	10 D
17. Stewart's P.C.P. formula	12.5	12.5 D
18. Bender-Seltzer polyantibiotic formula	10	10 D

Key to Symbols:
Numbers = Radius of zone of inhibition (in millimeters).
– = No zone of inhibition.
S = Zone of bacterial growth stimulation.
D = Zone of diffusion.

Pour plates were prepared for the application of disks saturated with the various drugs and antibiotics tested after the method of Waksman,[19] Scrivener,[20, 21] and others.

With brain-heart infusion agar employed as a growth medium, each plate was seeded with 0.5 ml. of inoculum. The inoculum was prepared from a broth culture obtained by pooling individual cultures of a series of ten carefully selected and controlled pulp and periapical infections.

Following the application of the "medicated disks" to the hardened agar, the pour plates were incubated at 34° C. The results were tabulated and photographed at twenty-four and seventy-two hours (Table I).

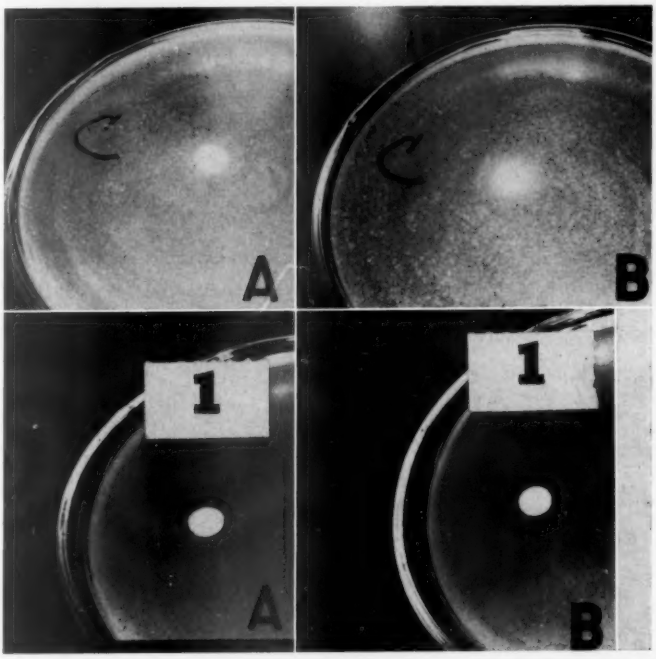

Fig. 1.—Top row: Control disk. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.
Bottom row: Oil of cloves with 25 per cent chlorobutanol without ointment base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Agents 1 through 5 were used as dispensed in their original containers in liquid form, and the disks were impregnated twenty-four hours prior to application to the hardened agar.

Agents 6 through 13 were prepared as follows:

30 drops of therapeutic agent

1 dram (teaspoonful) of water-in-oil emulsifying base (as noted in chart)

Fig. 2.

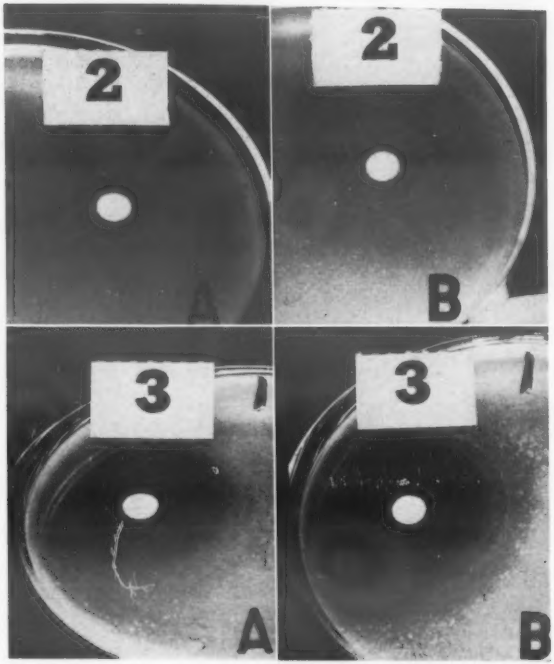

Fig. 3.

Fig. 4.

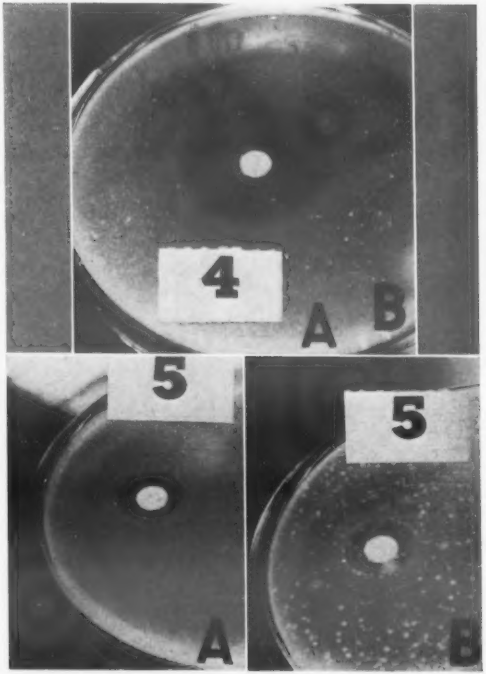

Fig. 5.

Fig. 2.—Camphorated paramonochlorophenol without ointment base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 3.—Formocresol without ointment base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 4.—Cresolated formaldehyde without ointment base. Incubation period seventy-two hours.

Fig. 5.—Beechwood creosote without ointment base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

The ingredients were spatulated under aseptic conditions and placed in sterile containers. The disks were impregnated for twenty-four hours prior to application to the hardened agar.

The terramycin-cortisone ointment (agent 14) was placed in a sterile container. The disk utilized as described previously.

The tetracycline (Achromycin) in ointment form (agent 15) was prepared as follows:

Tetracycline	250 mg.
Mineral oil	250 mg.
1 dram (teaspoonful) Aquaphor ointment	

Fig. 6.

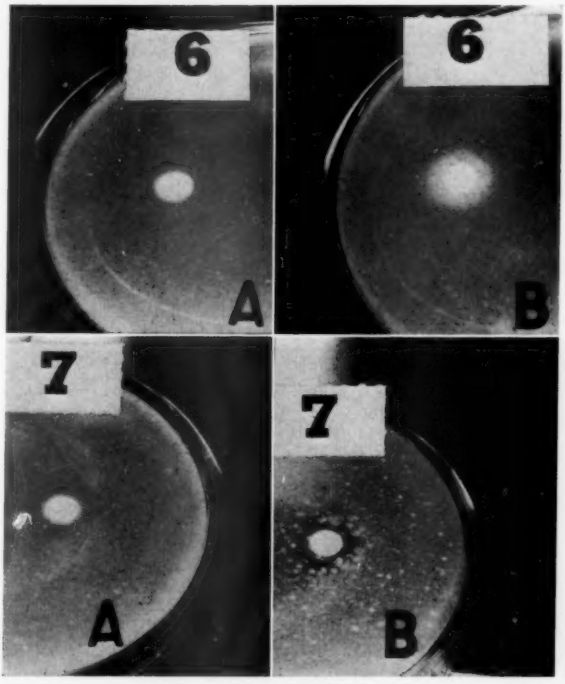

Fig. 7.

Fig. 6.—Oil of cloves with 25 per cent chlorobutanol with anhydrous lanolin base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 7.—Oil of cloves with 25 per cent chlorobutanol with Aquaphor base. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

The tetracycline and mineral oil were triturated in a sterile mortar. Aquaphor was added, and the mass was retriturated to a smooth consistency. The ointment was placed in suitable sterile container; disks were impregnated for twenty-four hours and applied to hardened agar as above.

Fig. 8.

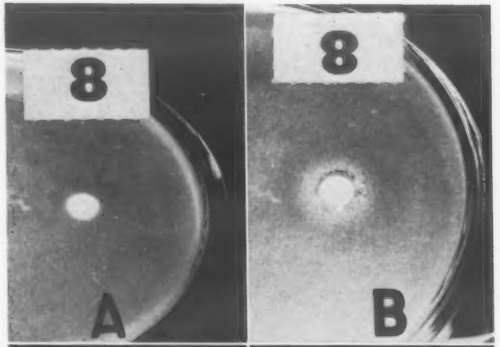

Fig. 9.

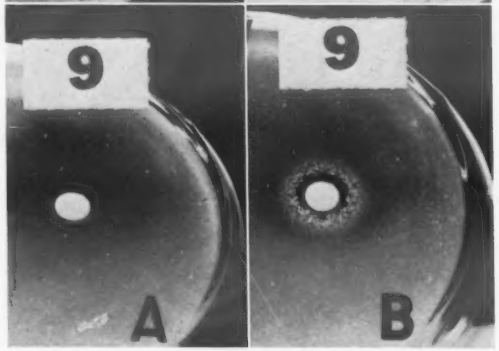

Fig. 10.

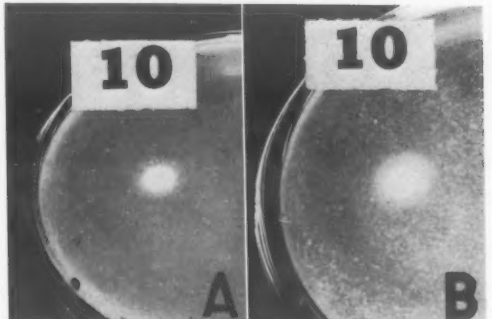

Fig. 11.

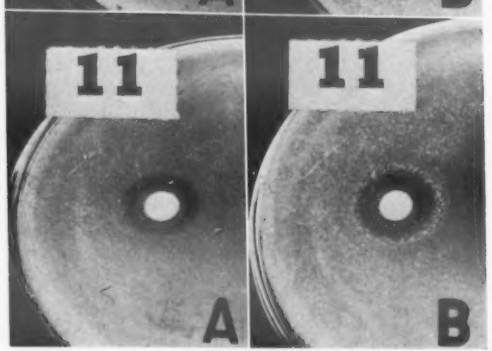

Fig. 8.—Camphorated paramonochlorophenol with anhydrous lanolin. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 9.—Camphorated paramonochlorophenol with Xylocaine ointment. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 10.—Camphorated paramonochlorophenol with Aquaphor. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 11.—Formocresol with Xylocaine ointment. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 12.

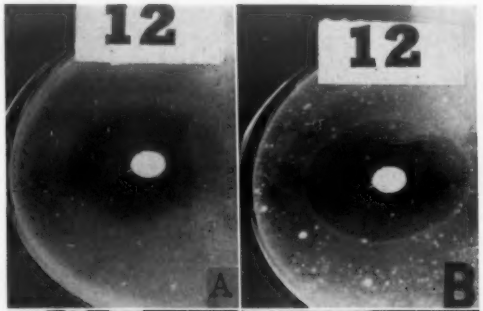

Fig. 13.

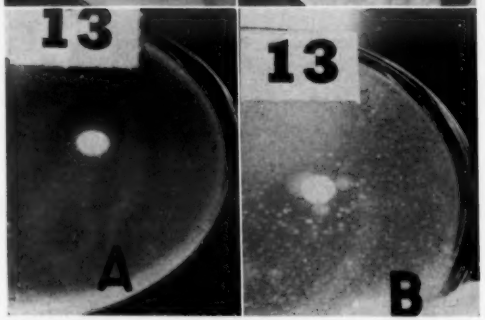

Fig. 14.

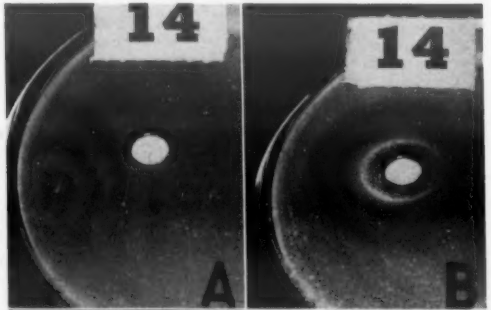

Fig. 15.

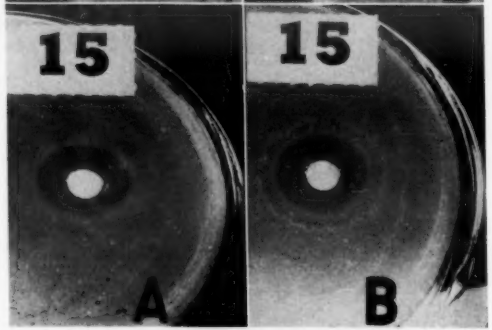

Fig. 12.—Formocresol with Aquaphor. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.
Fig. 13.—Beechwood creosote with Aquaphor. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.
Fig. 14.—Terramycin-cortisone ointment. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.
Fig. 15.—Tetracycline with Aquaphor. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Agents 16, 17, and 18 are the respective formulas of Grossman, Stewart, and Bender and Seltzer. These have been reported in the literature. They were utilized and applied as previously described.

Fig. 16.

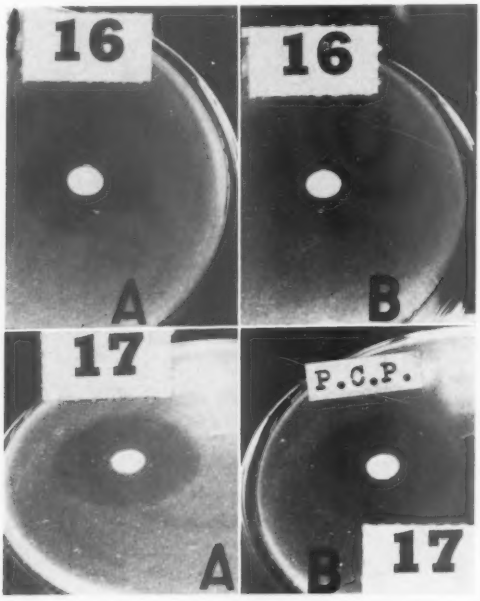

Fig. 17.

Fig. 16.—Grossman's PBSC formula. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

Fig. 17.—Stewart's P.C.P. formula. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

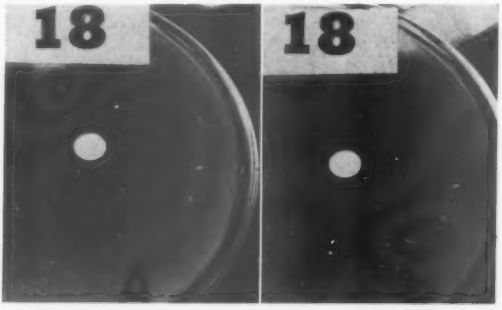

Fig. 18.—Bender-Seltzer polyantibiotic formula. *A*, Incubation period twenty-four hours. *B*, Incubation period seventy-two hours.

EXPERIMENTAL TABULATION

Table I and Figs. 1 to 18 represent the findings. It is to be noted that the radius of the "zone of inhibition" around each impregnated disk was measured in millimeters. Also note areas of bacterial growth stimulation.

Acknowledgment is made, with sincere thanks, to Eola Wooley, Ph.D., Biological Sciences, University of San Francisco, for her valuable assistance in the preparation of laboratory material.

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450 SUTTER ST.

RESEARCH

A HISTOPATHOLOGIC STUDY OF NERVE SCLEROSANTS*

Allen Jesse Koslin, D.D.S., Birmingham, Ala.

INTRODUCTION

THE aim of this study was to compare the histopathologic effects of absolute alcohol, Sylnasol,[1-3] and several other agents with potential local neurotoxic action, using the sciatic nerve of the rabbit as the test site. N/10 saline was selected as a control because it does not damage either muscle or nerve tissue.[4] Absolute alcohol has been employed as a neural sclerosant for approximately fifty-five years.[5] In recent years, 5 per cent sodium psylliate with 2 per cent benzyl alcohol (Sylnasol) has been used successfully as a nerve sclerosant and has been thought to be more self-limiting than absolute alcohol. Following the administration of calcium penicillin, sciatic nerve necrosis has been reported.[6] Supersaturated solutions of calcium hydroxide have been used for pulp-capping in clinical dentistry without destroying the dental pulp. Two per cent sodium dicetyl phosphate has produced intense fibroplastic reactions in animal experiments.[7]

METHOD

The agents tested were Sylnasol, 0.2 per cent sodium dicetyl phosphate suspension in olive oil, absolute alcohol, supersaturated solution of calcium hydroxide with a pH of 12.9, calcium penicillin 200,000 units per cubic centimeter with a pH of 6.2, and N/10 saline.

The "Dutch" breed rabbit was used as the experimental animal.

The fascial sheet covering the sciatic nerve was exposed, a 22-gauge needle on a 1 c.c. tuberculin syringe was passed through a small opening in the fascial sheet, and 0.25 c.c. of the desired agent was deposited with minimal pressure around the sciatic nerve. The site of deposition of the agents was the bifurcation of the sciatic nerve to form the peroneal and tibial nerves. The procedure was carried out in a manner designed to avoid mechanical irritation to the nerve.

From the University of Alabama School of Dentistry.

*A research project completed under the auspices of the Department of Oral Surgery, State University of Iowa.

Both sciatic nerves of each rabbit were utilized, with a different agent tested on each side.

Repeated needle pricks on the sole and dorsum of the foot, as a test for anesthesia, produced markedly inconsistent results. Clinical anesthesia was not practically demonstrable.

The animals were sacrificed one, two, three, four, six, eight, ten, and twelve weeks postoperatively. Two series of animals were used for each agent.

The thighs were biopsied and three sections, 4 mm. in thickness, were removed from each thigh as follows:

1. A proximal section near the head of the femur.
2. A central section at the site of deposition of the test agent.
3. A distal section near the distal end of the femur.

The specimen sections were stained with hematoxylin and eosin in accordance with accepted histopathologic techniques. One series for each agent was also stained with Luxol fast blue myelin stain.[8] The specimens were cut 7 microns in thickness, and microscopic slides were prepared. All slides were read at magnifications of ×28, ×100, and ×430.

RESULTS

All specimens in the series in which N/10 saline was the test agent showed no evidence of tissue damage or inflammatory response and were used as the controls (Figs. 1 and 2).

Sylnasol, in one-week specimens, produced a complete loss of axis cylinders and severe demyelinization of the nerve with no visible damage to the Schwann cells (Fig. 3). Muscle and fat damage was mild, with marked early muscle regeneration present. There was an exuberant early histiocytic and chronic inflammatory cell reaction without giant cells or phagocytosis (Fig. 4).

In the two-week specimens the intensity of the histiocytic reaction was reduced, the muscle and fat reaction was similar to the one-week specimens, and early granulation tissue was proliferating.

Complete nerve degeneration remained in the three-week specimens. The muscle and fat appeared to be almost normal. There was an abundance of giant cells with an otherwise scanty chronic inflammatory response.

The four-week specimens exhibited only focal nerve degeneration, most marked in the outer portions of the nerve fascicles. The adjacent tissues appeared to be entirely normal.

The six-, eight-, ten-, and twelve-week specimens appeared to be entirely normal in all respects, with the exception of one twelve-week series which showed a foreign body reaction and focal scarring in the perineural tissues. These latter findings may be attributed to greater initial damage or to an unknown injury sustained by the animal.

The agent did not exhibit a tendency to localize but, rather, spread along the fascial planes that were in association with the nerves.

Fig. 1.

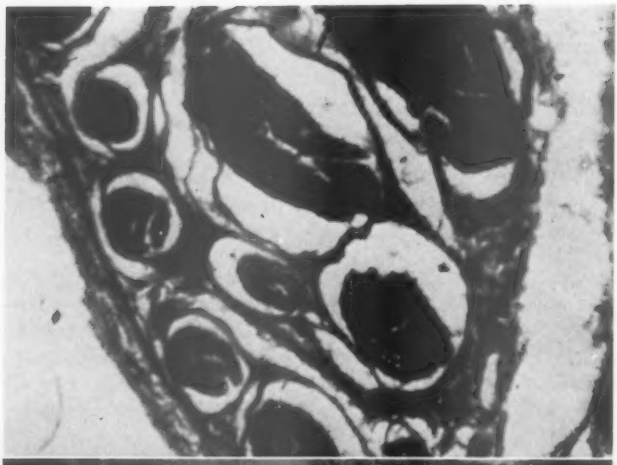

Fig. 2.

Fig. 1.—Photomicrograph of N/10 saline specimen at one week. Dense dark-staining nerves are the result of the uptake of stain by myelin and axis cylinders. (Luxol blue stain. Magnification, ×60; reduced ⅙.)

Fig. 2.—Photomicrograph of N/10 saline specimen at one week. There is no damage or tissue response present. (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅙.)

Fig. 3.

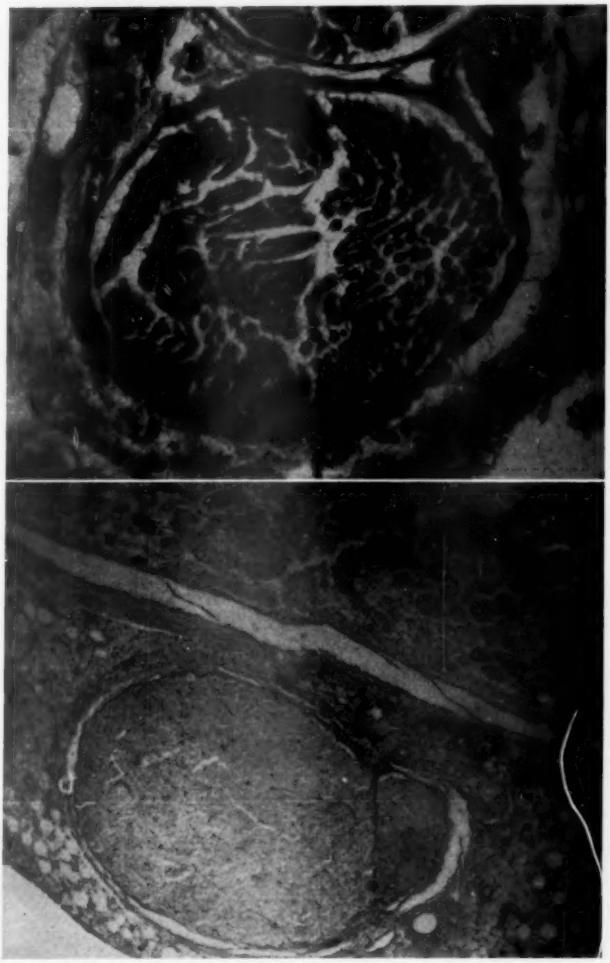

Fig. 4.

Fig. 3.—Photomicrograph of Sylnasol specimen at one week. Marked demyelinization is present, particularly in cross section. (Luxol blue stain. Magnification, ×120; reduced ⅛.)

Fig. 4.—Photomicrograph of Sylnasol specimen at one week. Exuberant histiocytic response and early marginal muscle regeneration are the most prominent features. (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅛.)

Sodium dicetyl phosphate produced no evidence of nerve damage in any of the specimens (Fig. 5). Minimal marginal muscle degeneration and regeneration were present in the one-, two-, and three-week specimens. Even in the one-week specimens, the central sections showed a massive large giant-cell reaction with an extensive chronic inflammatory response (Fig. 6). This

Fig. 5.

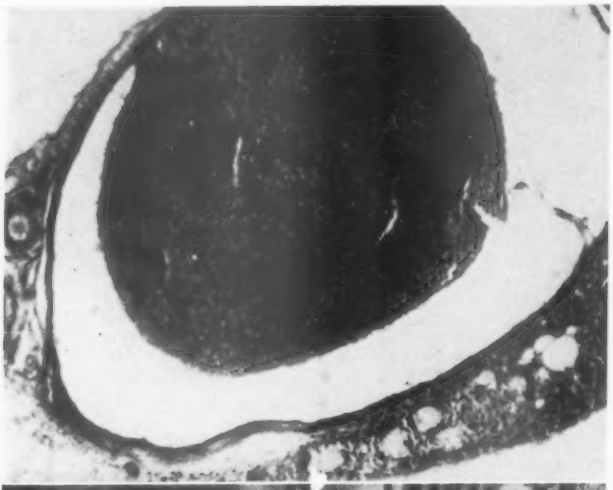

Fig. 6.

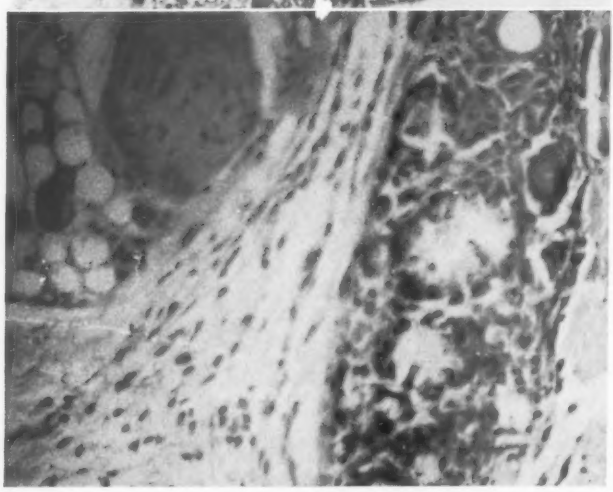

Fig. 5.—Photomicrograph of sodium dicetyl phosphate specimen at one week. Myelin and axis cylinders are normal. (Luxol blue stain. Magnification, ×60; reduced ⅙.)

Fig. 6.—Photomicrograph of sodium dicetyl phosphate specimen at one week. There are a marked histiocytic response, very large giant cells, and an abundance of fibroblasts. (Hematoxylin and eosin stain. Magnification, ×120; reduced ⅙.)

persisted, apparently unchanged, until the sixth week. The six-week specimens showed a diminution in the response. The chronic inflammatory cell proliferation was absent at ten weeks. The giant-cell reaction persisted through ten weeks but was absent in the twelve-week specimens.

Marked fibroblastic activity was present in the one-week specimens and continued through six weeks. Fibrosis was also apparent at one week, increased and matured through four weeks, and showed early cicatrization at six weeks. A moderate amount of cicatrix was present in the perineural fat and connective tissue in the twelve-week specimens.

The agent showed a strong tendency to localize, probably because of its viscosity, at the site of deposition.

Saturated calcium hydroxide solution, in the one-week specimens (Fig. 7), produced an outright focal coagulation necrosis without liquefaction of the nerve, affecting the axis cylinders, myelin sheath, and Schwann cells. These changes were severe. The agent showed a tendency to spread longitudinally along the fascial planes, producing a necrosis in the majority of the proximal sections. The distal sections, less directly affected, showed degeneration of the nerve with demyelinization, approximately fifty per cent loss of the axis cylinders and intact Schwann cells.

There were marked marginal muscle destruction and abundant early muscle regeneration. The fat and connective tissue had undergone an aseptic necrosis. An intense chronic inflammatory response, with many macrophages, separated the damaged from the normal tissues. There was an increase in nerve vascularity, as well as early organization of the necrotic tissue with early fibrosis (Fig. 8).

In the two-week specimens the nerve damage was equally severe, the chronic inflammatory proliferation was less intense, and the proliferation of granulation tissue was more abundant.

The three-week specimens were essentially similar to those at two weeks, with a slight increase in fibrosis and a questionable distortion of the perineurium.

The four-week specimens showed less nerve destruction in the proximal and distal sections, but at the site of injection it was equal to that of the preceding specimens. The perineurium was distorted and showed proliferation of granulation tissue. There was a general increase in the amount of granulation tissue, the fibrosis was maturing, and the scarring was intense. A granular precipitate of calcium was detectable, and giant cells were seen for the first time in this series.

The six-week specimens appeared to represent the beginning of a clean-up process. The nerve showed focal demyelinization, patchy loss, and swelling of axis cylinders and Schwann cells. The chronic inflammatory reaction was focal and minor in intensity. The granulation tissue response was lessened, and the scarring was increased. No further muscle destruction was evident, and many small young muscle fibers were present.

The eight-week specimens showed less demyelinization, very little axis cylinder loss, and intact perineurium, clumps of snarled Schwann cells, and axis cylinders running in haphazard arrangement with many entering into neighboring fascicles. The inflammatory response was absent, the scar shrinking and the muscles completely regenerated.

The ten-week specimens appeared to have complete healing, and all tissues were normal.

Fig. 7.

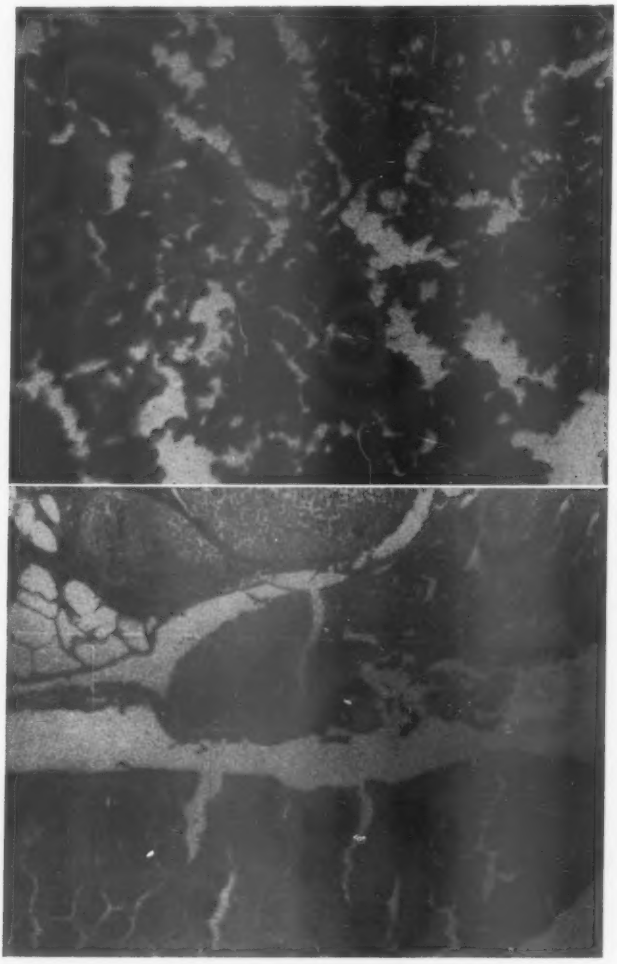

Fig. 8.

Fig. 7.—Photomicrograph of calcium hydroxide specimen at one week. There is marked demyelinization and a loss of approximately 50 per cent of the axis cylinders. (Luxol blue stain. Magnification, ×360; reduced ⅙.)

Fig. 8.—Photomicrograph of calcium hydroxide specimen at one week. An area demonstrating the severe muscle degeneration and necrosis. (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅙.)

The twelve-week specimens were similar to those at ten weeks with the exception of one series which more closely resembled the response at six to eight weeks, probably as a result of greater initial damage than in the other specimens.

No vessel damage or thrombosis was noted in this series.

Calcium penicillin produced no visible nerve changes in its entire series (Fig. 9).

Fig. 9.

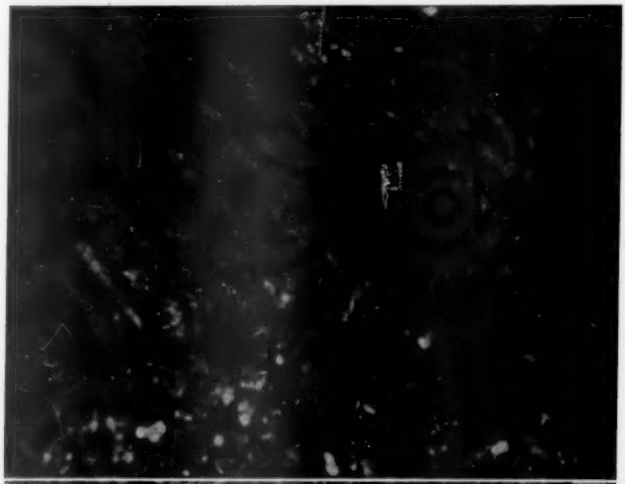

Fig. 10.

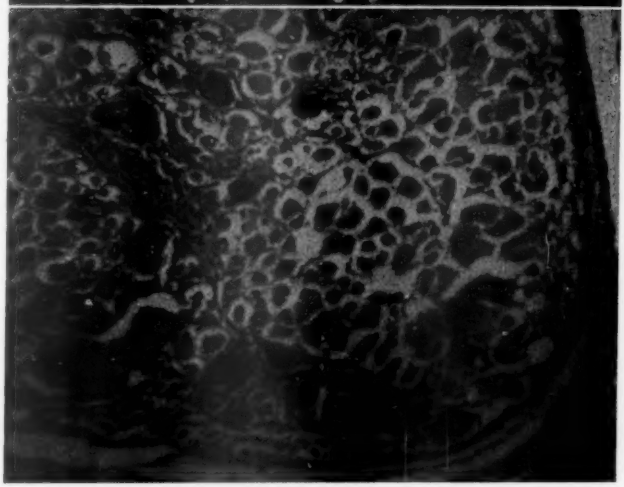

Fig. 9.—Photomicrograph of calcium penicillin specimen at one week. Nerve fibers are normal with intact myelin and axis cylinders. (Luxol blue stain. Magnification, ×360; reduced ⅙.)

Fig. 10.—Photomicrograph of calcium penicillin specimen at one week. Marked foreign body reaction with muscle destruction and calcium granules. (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅙.)

An intense foreign body reaction was elicited from the first (Fig. 10) to third weeks, and by the fourth week it was decreasing in intensity. The eight-week specimens showed no inflammatory response. Marginal muscle necrosis

Fig. 11.

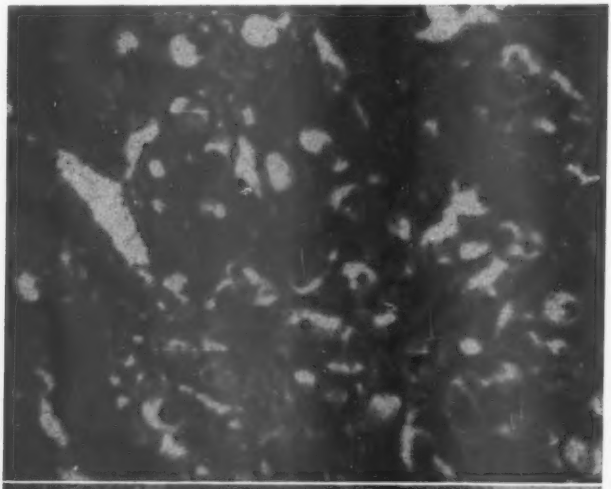

Fig. 12.

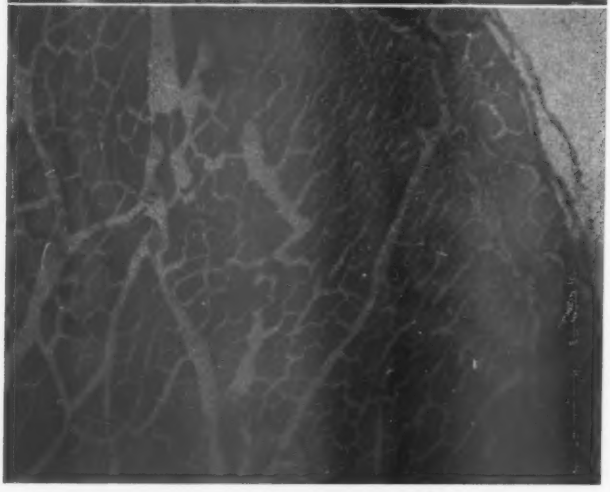

Fig. 11.—Photomicrograph of absolute alcohol specimen at one week. The nerve shows demyelinization and loss of axis cylinders. (Luxol blue stain. Magnification, ×360; reduced ⅙.)

Fig. 12.—Photomicrograph of absolute alcohol specimen at one week. There is marginal muscle destruction as well as a mild chronic inflammatory response separating the normal and damaged muscle. (Hematoxylin and eosin stain. Magnification, ×60; reduced ⅙.)

was present for two to three weeks, with some regeneration occurring. A marked fibroblastic response, beginning in the first week, produced large amounts of scarring by the fourth week.

The cicatrization, most pronounced around the nerves, persisted throughout the series.

Basophilic calcium granules, present by the second week, were found in the scars in all the later specimens and in greatest quantity immediately around the nerves.

The agent showed a marked tendency to localize at the site of deposition.

Absolute alcohol, in one-week specimens (Fig. 11), produced an outright coagulation necrosis of the nerve, similar to that resulting with calcium hydroxide. A mild chronic inflammatory reaction, some fat necrosis and a very minimal amount of muscle necrosis also resulted (Fig. 12).

The two-week specimens exhibited less necrosis and more degenerative changes in the nerves than in the one-week specimens. The axis cylinders were destroyed, and demyelinization was complete. The architecture was preserved, although swollen. There was some muscle regeneration as well as an increase in fibrosis at the site of deposition of the agent.

The three- and four-week specimens exhibited the same degenerative changes in the nerve as was seen at two weeks. The chronic inflammatory response had disappeared, minimal muscle regeneration persisted, and the degree of fibrosis (most marked at the central site) was moderate.

Approximately 50 per cent of the nerve fascicles in the six-week specimens showed degenerative changes. The adjacent tissues were normal with the exception of some slight fibrosis in the central sections.

The eight-, ten-, and twelve-week specimens were normal in all respects.

The agent did not localize, as evidenced by changes in the proximal sections similar in nature but not in degree to those at the site of deposition.

DISCUSSION

It may be assumed, from the absence of tissue reaction or damage in the N/10 saline series, that the operative procedure per se produced none of the changes demonstrable with the other test agents.

The degree of reaction to an agent at the site of deposition appears to be related, in part, to its tendency to pool at the site or to diffuse along the longitudinal fascial planes.

Sylnasol produced nerve degeneration of comparatively short duration and appeared to have a relatively more selective action on the nerves in comparison with its effect on the adjacent tissues. The clinical experience of those clinicians using Sylnasol for nerve blocks and the findings of this study are in contradiction to the conclusions drawn by Schultz.[3, 9]

Benzyl alcohol in 5 and 10 per cent concentrations has produced nerve and tissue damage.[10, 11] The effects of 2 per cent benzyl alcohol were not determined in this study. However, it should be considered as producing at least some of the damage and reaction that resulted with injection of Sylnasol.

Sodium dicetyl phosphate produced no demonstrable nerve changes. Its local fibroplastic properties are good.[7] The intense giant-cell proliferation would appear to be in response to the olive oil.[10]

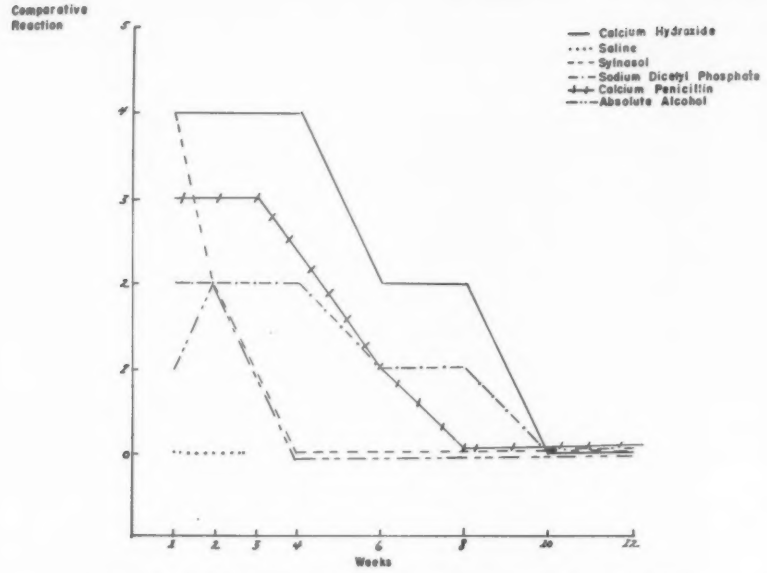

Chart 1.—Graphic representation of comparative reactions of tissues.

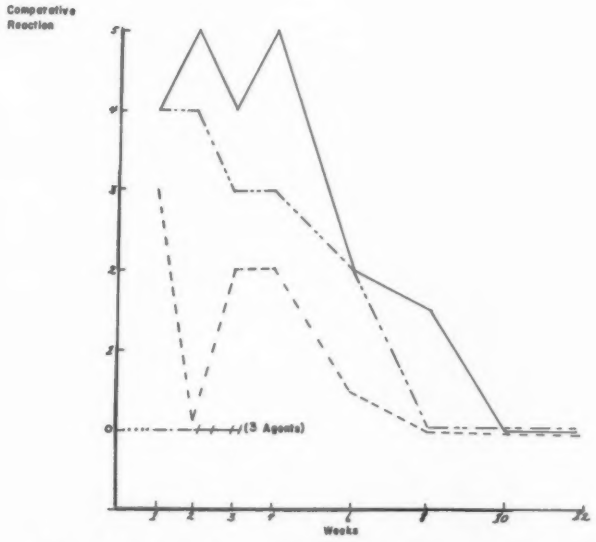

Chart 2.—Graphic representation of comparative reactions of nerves.

In all but one series, absolute alcohol produced degenerative rather than necrotic nerve changes, and an aseptic necrosis of fat and marginal musculature was seen in early specimens. The extent of degenerative changes produced by alcohol depends on the type of tissue injected. Skin and muscle tissues are not affected as severely as loose connective tissue or nerve tissue.[12]

"Strong alcohol" injected into a bundle of nerve fibers soon coagulates the albumin in the nerve sheath and fiber, resulting in degeneration of the nerve fibers.[13] In this study, absolute alcohol appeared to be the most highly specific agent for nerve tissue with the least amount of associated tissue reaction.

Absolute alcohol and Sylnasol had the same duration of damage to nerves.

Saturated calcium hydroxide solution produced nerve necrosis. The muscle and fat exhibited relatively greater destruction than the nerves. This is probably a result of the protection afforded the nerve by the perineurium. The distortion of the perineurium is indicative of nerve atrophy. The healing process with this agent was of interest in view of its tendency not to result in large amounts of cicatrization. The severity of the associated tissue reaction would probably contraindicate the use of this agent for clinical nerve blocks.

The results obtained with calcium penicillin are not in accord with those previously reported.[6] It is possible that the calcium granules and the dense scar around the nerves may produce functional changes in the nerves. On the basis of this study, calcium penicillin would not warrant clinical trial for nerve blocks.

CONCLUSION

This study indicates that the most effective agent, of those tested, for clinical nerve blocks is absolute alcohol.

Histopathologic changes in nerves are not necessarily a measure of the extent of functional changes which may occur. Nerves not damaged by an agent, as evaluated histopathologically, may undergo functional changes resulting in abnormalities of conduction.

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DIAGNOSIS AND TREATMENT PLANNING

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ORAL DIAGNOSIS AND CONSULTATION

*H. A. Brayshaw, Lieutenant Colonel, DC, USA**

TECHNICAL advances in dental materials, equipment, and procedures accomplished by diligent persons and co-ordinated subsidized research projects are tremendous. These discoveries often precede general practical application by many years. Narrowing this gap between technical scientific knowledge and clinical application is a constantly evolving educational process. Dentists alert to learn and to apply the improved aids become the most astute diagnosticians and therapists. The processes of study and evaluation are the instrumentalities of genuine professioal advancement.

Oral diagnosis in dentistry is often given passive and cursory attention. It is really one of the most important daily considerations that confront us. An accurate diagnosis is a necessity in the intelligent formulation of a treatment plan. Immediate palliative treatment for expressed complaints is frequently the only treatment given. If we are to give dental service that is worthy of dentistry's position as a scientific health service, careful, thorough diagnosis and treatment planning must be inaugurated. The importance of diagnosis is now emphasized by the American Dental Association's Council on Dental Education. That group requires that all approved intern, graduate, and residency training include instruction by a qualified diagnostician. "The patient and the dentist benefit mutually from the time devoted to a comprehensive diagnosis."[1]

Special training in oral diagnosis is becoming available as an integral part of a well-balanced advanced training curriculum. To become an astute diagnostician, one must have a great deal of knowledge and repeated experience. An accumulation of experiences can be presented in a teaching situation, but these become more meaningful and more valuable as they become a personal challenge and are solved by the individual dentist. The ability to recall at the crucial time and, to use the knowledge gained is a mental discipline acquired by assiduous study and keen observation. Kolmer[2] stated: "Unfortunately it frequently is difficult and sometimes impossible for physicians

*Chief of Periodontia, Madigan Army Hospital, Tacoma, Washington.

and dentists to detect potential primary foci of infection. Since the detection depends largely on individual diagnostic skill and the methods employed, the incidence of detection will vary a great deal." Obviously, much can be gained from an exchange and comparison of ideas in a consultation situation.

In becoming an outstanding professional service, dentistry has suffered many growing pains. Notably, there is a predominant generosity among educators and clinicians who unselfishly contribute and exchange ideas and experiences to improve the oral health ministrations. We must overcome any reluctance to confer or exchange ideas on treatment and diagnosis with fellow dentists. Hospital practice investigations of medical service reveal that the record of success is directly proportional to the number of consultations employed. Hospital records show that 20 to 50 per cent of cases involve consultation. Letourneau and associates[3] write: "Successful consultation requires (1) a recognition of and mutual respect for individual talents and knowledge, (2) therapy is determined by agreement and utilization of each plan as it complements the other." Mortality and morbidity rates drop as consultations are more freely used. A mandatory consultation is required in all cases involving grave surgical procedures that could seriously modify the function and appearance of the patient or result in his demise. We could profitably adopt a code of diagnostic investigation that would immeasurably improve the practice and prestige of the dental profession. Obviously, the consultation is not always convenient or available to the patient's economic position. The dentist has an ethical obligation to exercise utmost diagnostic acumen to arrive at the best diagnosis and treatment plan that he can devise. Fortunately, most dentists have had excellent training and are capable of making good decisions for treatment in most cases. Unusual and involved problems do arise, however. We have developed specialty fields in which the specialist is outstandingly qualified to provide aid and advice in the complicated problems. The specialties of periodontia, oral pathology, prosthodontics, oral surgery, and orthodontics have become recognized as essential. These have examining board requirements to test and certify specialists as to their qualifications. This assures the profession and the public that dentistry is a respected, mature component of the health services.

APPROACH TO DIAGNOSIS

What is the process of making a diagnosis? We gain impressions that lead us to suppositions. We must seek as many informative impressions as possible in a searching examination and history to aid in arriving at a diagnosis. The medical and dental histories, carefully taken, frequently reveal the true nature of the complaint. Current and recent medications and therapy are of special significance. It is important to be a good listener and enable the patient to relate the pertinent data. Repeated questioning at successive visits may be required to bring out key facts. First, we obtain a provisional or working diagnosis. It may also be considered a presumptive diagnosis. This is the most obvious and initial diagnosis which will suffice until a change

in diagnosis is indicated. The immediately urgent problem or emergency condition should be identified, evaluated, and treated at the outset. This will at least give the patient relief and comfort and will restore composure. Examination may reveal a problem that is more severe and more grave than that which is recognized by the patient. This should then be studied, the patient should be advised, and treatment should be instituted. No major clinical problem can be resolved unless the cause can be found and treated. Palliative treatment is very necessary, but it should be considered a temporary expedient. We must detect and correct any injurious habits that are observed. Prophylactic procedures must be instituted, and the patient should be shown how to help himself by daily personal hygiene. Thus, we may prevent regression or the introduction of additional complications. The differential diagnosis is the one that we choose by listing the many similar likely diagnoses. Through a process of elimination, we select the diagnosis which fits most fully the clinical problem under consideration. This differential or clinical diagnosis is not necessarily the correct one.

One must not hasten to conclude that there is no trouble if the cause of the patient's complaint or fear is not obvious. Most dental patients rally great courage or are driven by discomfort and pain to see a dentist. It is wrong to give them false assurance that all is well. Impressions, suppositions, guesses, and observations are processes of thought in seeking a diagnosis, and no one of them should be mistaken for the diagnosis. For instance, the radiograph gives impressions that can be interpreted, but there is truly no "x-ray diagnosis." This is only an opinion of limited fact. Much more investigation is required. We should not yield to the temptation to use a high-sounding word as the diagnosis when it is only a supposition of the type of ailment.

In order to get an accurate diagnosis, a number of clinical and laboratory tests must be employed. We should request the indicated laboratory tests, such as bacteriology, hematology, tissue pathology, urinalysis, blood chemistry, and endocrine evaluations. The x-ray, thermal, electric, palpation, percussion, probing, and functional observations should be employed. Subjective data and history contribute to the pertinent inquiry. When these are fully evaluated to compose the big picture, we may obtain an accurate diagnosis. This may be a different diagnosis, or it may be the same as the provisional diagnosis. If it is the same, it is known as a confirmatory diagnosis. However, the patient may have other ailments that could provide additional diagnoses which either do or do not influence the principal problem in question. Hence, we gather more known diagnostic facts upon which to formulate a treatment plan.

After treatment and resolution of the clinical problem, we review the case progress and correlate it with the therapy, so that we can arrive at a final diagnosis. It may be the same or it may vary from any of the previous diagnoses. We might now agree that a diagnosis is a label or term applied to a clinical entity which implies its recognition determined by the processes of seeing, probing, testing, recording, visualizing, thinking, summarizing, and concluding.

DIAGNOSTIC ACUMEN

To become an astute diagnostician, one must have vast training, experience, and self-discipline to observe keenly and evaluate accurately. Patients often present a garbled, confused, incoherent account of their problems. "In seeking to help people even in very simple situations," Garrett[4] writes, "we need to listen not only to their objective requests but also to the undertones which reveal their feelings and give us clues as to perhaps even more serious objective situations not overtly revealed." It is by careful questioning to elicit the true facts of the problem that the fundamental cause can be determined. One should ask clear-cut questions that merit direct answers, so that the patient will not reply with unrelated statements. The cause foremost in the patient's mind might be the current feature, but that is often a late manifestation in the profusion of symptoms accruing as the cycle of a disease process progresses. Tact must be exercised to put the patient at ease so that pertinent information is readily forthcoming. Skill in diagnosis and technique is signally important, for this gives both the doctor and the patient a magnetic feeling of confidence. We observe only the phenomena that we are mentally alert and trained to see. Consequently, it behooves us all to become better prepared. Humility is a quality of personality that endears one to others and eliminates obstacles to good human relations. If the dentist is humble, the patient will feel the freedom so necessary to overcome fears that often cause professional failure. We should constantly strive to improve technical clinical skills and scientfic knowledge by maintaining academic enthusiasm. Human understanding of mortal problems will foster our approach to each patient as an individual and not as just another case number in a long and tedious day in the clinic. We must never indicate to the patient that he is just another burdensome problem. We should have courage in our convictions so that we can clearly and affirmatively state our opinions, which we are prepared to substantiate by fact and statistics.

CONSULTATION

Consultation may be necessary. Supportive care must not be neglected while investigation is in progress. We should enlist the aid and counsel of our confreres in the medical professions to improve the patient's general health. Miller[5] wrote: "Of equal import to the dental practitioner is the fact that systemic conditions, unrecognized, will unduly prolong local treatment or invite complete failure." We should enable the patient to become stronger to combat the oral problems. Now we can advance with a planned order of disease management and indulge in the intricacies of the selected reconstruction procedures.

Consultation includes a formal deliberation on findings, diagnosis, and prognosis of a case. It should take up the advisable therapeutic measures. Consultations should be written and should become a part of the patient's record. They are valuable to the patient and to the doctors. They may have

legal significance in the event the case comes into litigation. Records often are required in court proceedings to show that proper efforts in diagnosis, consultation, and treatment were exercised. Verbal consultation or agreements would only constitute hearsay. As we more frequently accept and contribute ideas on diagnostic problems, we enrich our professional lives.

CONSULTATORY STIPULATIONS OR REQUIREMENTS

To accomplish most from the consultation, it is best to have a mutual understanding and acceptance of factors that favor this type of communication. Let us review some of these factors and adopt adjustments where needed. Petty jealousies or grievances must be discarded in the interest of mastering difficult clinical problems. The patient's welfare is to be placed above any individual aversions or personality quirks. The self-confident dentist feels that he alone should and can solve all clinical problems and is prone to be too proud to seek assistance. No man knows all, and few know all about one facet of science. Dentists who have qualified as specialists in the recognized specialties should be especially capable of aiding in diagnosis and counsel on unusual cases. This is an adjunct that should be utilized by the general practitioner and between specialists. Unfortunately, this idealism is occasionally impeded by attitudes of self-love. Pet opinions often are so dear to the self-centered that these persons cannot risk exposure to consultation. Vanity which isolates one is a great hindrance.

A careful, common-sense, over-all evaluation of the patient, as well as a provisional diagnosis, should be made by the referring dentist and should be included on the consultation request. Some effort should be made to solve the problem. It may be surprising to find that the solution often becomes obvious and consultation is then unnecessary. Pompous, dogmatic attitudes must be abandoned, for they are a deterrent to good relationships and they prevent communication. Fear, confusion, hesitancy, embarrassment, resentment, and anger result from high-handed relationships and most certainly discourage cooperation. The specialist of great professional prestige should be wise, tolerant, and considerate instead of assuming the figurative position of a "tin god." There should be a mutual respect between the referring agency and the consultant. Each should be ready to compliment the other on his diagnostic acuity and good judgment in prescription of therapy.

The therapy is best determined by agreement and fusion of contributory ideas or at times by specific recommendations based on scientific fact and practical experience. Preferably, the consultant should be unrelated, either genetically or in a business partnership. More amicable results are generally obtained if one can get an impartial and unbiased opinion. The patient becomes more confident and is not likely to suspect collusion. Patients are entitled to the assurance and reassurance which a consultation can provide. They have a right to express frank doubt and to ask for another opinion on their problems. Skills and knowledge do vary greatly with the dentist's likes,

dislikes, and favorite types of dental therapy. Each specialist is keenly observant of his particular field and must necessarily focus his energies and time on them. This tends to narrow interests to the point of unawareness of other problems. The necessity and value of consultation between those who excel in specialized fields therefore become evident.

The consultant and the referring dentist are entitled to the courtesies of the "Golden Rule." Neither should be insulted or slighted. Proper representation of the complexity of the problem should be made. No patient should be sent for consultation with the impression or assurance that this is a simple problem for the consultant. It should not be said that the case is referred only because time and equipment are not adequate in the referring office. We should not oversimplify the referred problem. It is better to make a factual statement. This will create greater respect for the dentist and give the consultant a fair start. The data on general health, dental findings, and current treatments along with roentgenograms, models, and diagnostic charts should accompany the patient; this saves a great deal of duplication of effort. A statement should be made on the request as to whether the patient is being sent for diagnosis, for therapy prescription, or for treatment and report.

CONSULTATION VALUES

The advisory value utilizes the consultant's vast wealth of knowledge and experience to recommend correct treatment and possibly shorten the period of illness. Humility, properly displayed, inspires confidence. "To refer a case for consultation is often evidence of good judgment for which the patient gains greater respect for the referring dentist."[6] It is a wise man who realizes when a problem exceeds the limitations of his knowledge and ability. The consultant's knowledge, recalled to solve the patient's dilemma, expedites resolution and gives protection to all concerned. There is a molding and/or fusing of considered opinions. This is a deterrent to poor practice. Slip-shod palliative procedures become minimized, and truly therapeutic and remedial treatment is administered. Better treatment planning ensures a superior result. Consultation provides the opportunity for the dentist and his profession to gain stature immeasurably. Utilization of counsel helps us to share responsibility. No man is infallible. He may inadvertently overlook some minor point that could prove to be a most important factor in obtaining results. The consultation provides documented reports that cumulatively aid in statistically analyzing results. These help to shape concepts on an improved treatment regimen. This is an excellent teaching medium and is essential in the intern, graduate, and residency training programs.

POTENTIAL WEAKNESSES IN CONSULTATION

The consultation occasionally has complications and shortcomings. We should take an objective view of these and attempt to avoid or to obviate their recurrence. Better liaison and proper initiation of the request could nullify most of the objectionable features. In the maze of referrals, the patients are

buffeted around too much. They do not find out the nature of their ills. Fitzgerald[7] says: "The patient grows more and more unhappy as he is shuttled from specialist to specialist without getting a good picture of what is wrong with him or a notion of how to set it right." This consumes much valuable time. Treatment might be delayed. Delay allows the condition to become more serious.

Without properly prepared consultation or referral requests, too many tests and treatments are duplicated or possibly contradicted. Patients are submitted to unnecessary expense. Frequently, the responsibility for consummation of treatment is lost or forgotten. A specific responsibility for completion of treatment and review of findings must be agreed upon or stated in the requests. Contradictory statements of opinion on diagnosis and treatment should be discussed between the doctors and not in the presence of the patient. Confusion and doubt are aroused if the patient listens to discussions which he cannot evaluate. The patient might give up in confusion and feel that no one knows how to help or cares to administer definitive treatment. Since patients are submitted to great inconvenience and expense to obtain consultation, they should be given a maximum of prompt attention. However, the consultant should supply only advice unless the patient is referred for treatment. Optional treatment should be instituted only upon the concurrence of the referring dentist and the patient.

REASON FOR REQUEST

A thorough general diagnostic evaluation is one objective desired when a patient is referred to a clinic of diagnostic specialists. On occasion, specific information about a particular problem is sought from the prosthodontist, the periodontist, the orthodontist, the restorative specialist, the oral pathologist, or the oral surgeon. This should be clearly stated. All reports should be made promptly in writing. A telephone report is justified only in an emergency, and it should always be substantiated by a written report. Prompt, efficient, accurate reports expedite case management. Progress and final reports should be made on involved cases requiring prolonged treatment by the specialist.

INTERVIEW WITH THE PATIENT

After scrutinizing examinations, consultations, and evaluation of findings, the patient is most curious and is entitled to know the course that is recommended for him. A brief résumé of findings and prognosis, devoid of technical details, should be provided to give reassurance if possible. Prognosis is an appraisal of acquired data which projects the likely duration, course, and termination of a disease or injury. Our storehouse of experience affords the judgment to anticipate the course and duration of treatment of oral pathologic processes. The patient is often comforted to know what to expect. When the prognosis is doubtful or poor, the advisability of informing the patient should be considered, and if he is to be informed this should be graciously

accomplished. If necessary, the bad news should be given to close relatives and dependents so that the indicated adjustments can be made. During the patient's orientation the conferring agencies should be complimented for detailed study and consultation. Astute observations in the initial examination should be cited. The gravity of the situation should be reviewed to emphasize the good judgment shown by our confreres. In the explanations, simple language, diagrams, gestures, and illustrations, fully understandable to the patient, are imperative. One should be sure that the patient understands the terminology. Understanding gives patients courage and confidence that they are in capable hands; professional jargon only confuses them. Specialists must guard against using language that is peculiar to their group but possibly vague to the general dentist.

The effects of lack of treatment should be explained. The time factors in treatment often make it necessary to alter a plan to meet the availability of the patient. Explanation of the stages and steps in the treatment plan prevents misunderstanding as to the scope, timing, and completion of treatment. Economic and practical values should be fully considered and presented for the patient's decision. He is in the best position to decide if it is opportune or if he is monetarily prepared to select an ambitious dental program. The dentist must not decide this for him but must be prepared to offer alternate acceptable plans beneficial to the patient which he is financially able to manage. The patient's attitude toward treatment is supremely important. "Treatment planning, when employed," says Adams[8] "ties all these elements together and provides the dentist with the practical knowledge necessary for success and satisfaction in practice."

If the therapy is urgent and mandatory, the patient should be apprised and directed. If it is optional, however, he should be informed of benefits to be derived and afforded a chance to make his own decision. If the facts of the clinical findings and the therapy prescriptions are well presented, the patient's decision will be to follow the dentist's recommendations with enthusiasm and satisfaction.

Some clinical problems appear to have insurmountable complications, confusion, and obscurities. For these we have no known tests for identification of causes and no specific therapy. At such times, we must frankly realize there are influences beyond the present scope of human knowledge and that often these can be resolved only by devout spiritual faith in the power of one's religion. Sir William Osler once said: "Nothing in life is more wonderful than faith—the one great moving force we can neither weigh in the balance nor test in the crucible."

SUMMARY

The importance and scope of investigations required for adequate diagnosis and consultation are presented. The advantages and pitfalls of consultations, with suggestions for avoiding the disadvantages, have been considered. The justification and procedure in the interview with the patient are emphasized.

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PROFESSIONAL NEWS ITEMS

University of Alabama

The School of Dentistry announces the following refresher courses:

Dental Assisting. Feb. 14 and 15, 1959.
Gertrude M. Sinnett, B.S., D.H.

A Practical Approach to the Full Denture Problem. Feb. 21-23, 1959.
Irving R. Hardy, D.M.D.

Removable Orthodontic Appliance Construction. Feb. 28—March 2, 1959.
Samuel D. Gore, D.D.S. (Course limited to practicing orthodontists.)

For further information, please write to Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Ave. South, Birmingham, Alabama.

Boston University

The Department of Stomatology, Division of Graduate Studies, announces the following postgraduate courses:

Advanced Course in Periodontal Therapy. March 2-7, 1959. Henry M. Goldman, Bernard S. Chaikin, and staff.

Techniques in the General Practice of Dentistry. March 9-14, 1959. Staff.

Periodontal Therapy. March 16-27, 1959. Henry M. Goldman, Bernard S. Chaikin, and staff.

Tooth Preparation and Elastic Impression Material Techniques in Restorative Dentistry. March 30—April 1, 1959. Arthur Kahn.

Full Denture Prosthesis. April 6-15, 1959. Chester Landy.

Occlusal Adjustment. April 27—May 1, 1959. Bernard S. Chaikin, Henry M. Goldman, and staff.

Endodontics. May 11-15, 1959. Herbert Schilder.

Periodontal Prosthesis. May 18-22, 1959. Lee Talkov and David J. Baraban.

Surgical Technics Employed in Periodontal Therapy. May 25-29, 1959. Lewis Fox.

Pulp Protection in Operative Dentistry. June 4-6, 1959. Harold Berk.

Periodontal Pathology. June 8-12, 1959. Henry M. Goldman, Jack Bloom, and staff.

For further information, write to Director of Postgraduate Studies, Department of Stomatology, Massachusetts Memorial Hospital, 750 Harrison Ave., Boston 18, Massachusetts.

Baltimore College of Dental Surgery

The Baltimore College of Dental Surgery, Dental School, University of Maryland, announces a course in endodontics for the general practitioner. The course, which will be under the direction of Dr. Irving I. Abramson, will be conducted between 9 A.M. and 5 P.M. daily, Jan. 11-16, 1959.

Tuition is $250.00. Each participant will receive a kit containing root canal instruments. Enrollment is limited to ten.

Eastman Dental Dispensary

The Eastman Dental Dispensary, Rochester, New York, announces twenty internships and clinical fellowships, providing preparation for general practice or dentistry for children. Elective part-time assignments are offered in periodontics and also in the dental clinics of the Strong Memorial and Genesee Hospitals and in the Dispensary Research Laboratories.

Appointments, paying $190.00 to $290.00 per month, are from September to June. Continuing second- and third-year appointments are available in orthodontics, periodontics, pedodontics, and research. These may be combined with a M.S. degree program.

Inquiry should be directed to Basil G. Bibby, D.M.D., Director.

University of Illinois

A postgraduate refresher course in the use of premedicating agents and general anesthesia is offered Feb. 12 and 13, 1959, following the Midwinter Meeting of the Chicago Dental Society.

The course will be directed by Dr. Elaine Stuebner, Department of Oral and Maxillofacial Surgery. The faculty will include Drs. A. G. Anderson, head of the Department of Oral and Maxillofacial Surgery; Reuben C. Balagot, assistant head of the Division of Anesthesiology; Max S. Sadove, head of the Division of Anesthesiology; and John M. Spence, head of the Department of Operative Dentistry.

Further information can be obtained from the Division of Postgraduate Education, University of Illinois College of Dentistry, 808 South Wood St., Chicago 12, Illinois.

University of Kansas City School of Dentistry

Applications for graduate study are now being accepted for a three-year course in oral surgery offered in conjunction with Kansas City General Hospitals and the Kansas City Veterans Administration Hospital. These programs are designed to meet the requirements of the American Board of Oral Surgery.

All graduate courses begin July 1. Candidates for the oral surgery program should have their applications on file not later than Jan. 1, 1959. Inquiries should be sent to the Director of Graduate Training at the University of Kansas City School of Dentistry.

The School also announces that two rotating internships are offered in conjunction with the Veterans Administration Hospital of Kansas City, Missouri, and the Veterans Administration Hospital of Wadsworth, Kansas. Information regarding these internships may be obtained by writing to James P. Darnell, Chief of the Dental Section, Veterans Administration Hospital, Kansas City, Missouri, or to Dayton D. Krajicek, Chief of the Dental Section, Veterans Administration Hospital, Wadsworth, Kansas.

University of Kansas City School of Dentistry

An expansion of the cooperative program of the University of Kansas City and the Veterans Administration Hospital has been announced by Dr. Hamilton B. G. Robinson, dean of the School of Dentistry, and Dr. James P. Darnell, chief of dental services for the hospital.

The cooperative program, begun five years ago, now also includes a residency in oral surgery at the hospital for two dental graduates, a rotating internship for one graduate student each year, and mutual consultation services between the hospital and school staffs.

In the rotating internship plan, the dental intern spends a year at the hospital, devoting 80 per cent of his time to the hospital and 20 per cent to the Dental School for postgraduate study.

The residency program in oral surgery is under the direction of Dr. Darnell and Drs. Charles J. Brown and Robert E. Allen of the Dental School. Two graduates who have completed a full year of graduate education at the Dental School are selected annually as residents and spend two additional years at the hospital, specializing in dental surgery. Residents are thus qualified for the specialty board examination in oral surgery and at the same time have qualified to receive the master's degree.

University of Minnesota

A course in advanced oral pathology will be given April 20 to 24, 1959. The program follows:

Monday, April 20

Non-neoplastic Diseases of the Jaws. W. Shafer.
Neoplasms of Bone. D. Dahlin.
Tumors of the Neck. R. Marcial-Rojas.

Tuesday, April 21

Benign Salivary Gland Tumors. A. P. Chaudhry.
Malignant Salivary Gland Tumors. A. P. Chaudhry.
Oral Syndromes. R. J. Gorlin.

Wednesday, April 22

Odontogenic Tumors. R. J. Gorlin.
Soft Tissue Tumors. R. Lattes.

Thursday, April 23

Radiation Therapy of Oral Neoplasms. D. Mosser.
Roentgenology of the Jaws. E. Stafne.
Lymphomas and Allied Lesions. E. Soule.
Roentgenology of the Head and Neck. H. Peterson.

Friday, April 24

The Dermatoses. R. Goltz.
Benign and Malignant Skin Tumors. H. Lund.

The tuition fee will be $65.00. For further information, write Dr. R. J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis, Minnesota.

University of Pennsylvania

The following postgraduate courses are announced by the School of Dentistry:

The Mandibular Third Molar. Feb. 9-13, 1959. P. Philip Gross and associates.
Oral Medicine. March 9-13, 1959. Lester W. Burket, S. Leonard Rosenthal, and associates. (Under the joint sponsorship of the University of Pennsylvania and Temple University.)
Oral Diagnosis and Roentgenology. Feb. 16-20, 1959. Lester W. Burket, LeRoy M. Ennis, and associates.
Partial Dentures. Feb. 23-27, 1959. M. M. DeVan and associates.
Occluso-Rehabilitation—Principles of Diagnosis and Treatment. March 2-6, 1959. Louis Alexander Cohn.
Principles of Occlusion. March 4-6, 1959. Lewis Fox.
Applied Anatomy. March 16-18, 1959. Harry S. Sicher.

For further information, please write Dr. Lester W. Burket, Dean, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

Michael Reese Hospital

A course entitled "The Medical Laboratory in Clinical Dentistry" will be presented Feb. 12 and 13, 1959, immediately following the Chicago Mid-Winter Meeting.

Dr. Emanuel Cheraskin, Professor and Head of Department on Oral Medicine, University of Alabama, will present the physiology and pathology of blood and urine, tests for infectious diseases, chemical intoxication, neoplasia, stress syndrome, nutrition, and hormonal deficiencies and illustrate how they may be utilized in the practice of dentistry. Dr. Arthur Elfenbaum, Professor Emeritus, University of Illinois and Northwestern University, in charge of Diagnostic Division, Department of Dentistry, Michael Reese Hospital, will correlate Dr. Cheraskin's presentations to diagnosis and x-ray interpretation in the dental office.

The fee for the two-day course will be $35.00.

For further information and the detailed program, write to Dental Department, Michael Reese Hospital, 29th St. and Ellis Ave., Chicago 16, Illinois.

City of New York Department of Hospitals

The Dental and Oral Surgery Service of Cumberland Hospital, Brooklyn, New York, is accepting applications for internships for the calendar year starting July, 1959. Application forms may be secured by writing the Medical Superintendent, The City of New York Department of Hospitals, Cumberland Hospital, 39 Auburn Pl., Brooklyn 5, New York.

Veterans Administration Medical Teaching Group Hospital

The Veterans Administration Medical Teaching Group Hospital of Memphis, Tennessee, announces that one rotating dental internship and one residency in prosthodontia will be available for the year beginning July, 1959.

Information and application forms may be obtained from the Chief, Dental Service, Veterans Administration Medical Teaching Group Hospital, Memphis, Tennessee.

ABSTRACTS OF CURRENT LITERATURE

ORAL PATHOLOGY

Primary Hepatoma With Metastasis to the Mandible. A. Dick, S. G. Mead, M. Mensh, and W. E. Schatten. Am. J. Surg. 94: 846, December, 1957.

This case of primary hepatoma with metastasis to the mandible is reported as being without precedent in the literature.

A 57-year-old white man had a third molar removed from the mandible two and one-half years previously and severe complications followed.

One of the authors found an elevated spongy mass on the crest of the mandibular ridge, extending from the second premolar area to the ascending ramus; the area was edentulous. There were no palpable masses in the neck, submental, or submaxillary areas. X-ray examination revealed that the mandible was involved.

An excision biopsy reported "malignant tumor," and the pathologist commented that it looked like a "liver biopsy."

An exploratory laparotomy was done, and the liver was found to be diffusely modular.

The pathologist's report was hepatoma.

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ORAL MEDICINE

Trigeminal Neuralgia: Special Consideration of Nonsurgical Treatment. J. G. Rushton and H. N. A. MacDonald, J. A. M. A. 165: 437, October, 1957.

The authors have studied the cases of 155 patients with trigeminal neuralgia, seen in the Mayo Clinic during 1953, in an attempt to learn something of the natural history of the disease. Whereas the disease was found to be primarily one of the middle or later years of life, nineteen of the 155 patients noted symptoms before the age of 40 years. From the study of the length of history, it would appear that spontaneous cures may occur but that such occurrences are very rare. Seven patients suffered bilateral trigeminal neuralgia, with an interval between the onset of pain in the two sides of from three to twenty-four years. One patient suffered both glossopharyngeal neuralgia and trigeminal neuralgia.

The time between the onset of illness and the presentation of the patient for his first treatment plus the duration of longest spontaneous remission will give some clue as to the expected future course of the illness. The patients who have had trigeminal neuralgia for a long time and who have enjoyed long remissions may find that their disease follows a somewhat benign course.

T. J. C.

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PUBLISHED BY
THE C. V. MOSBY COMPANY
ST. LOUIS 3, MO., U.S.A.

Official Publication of

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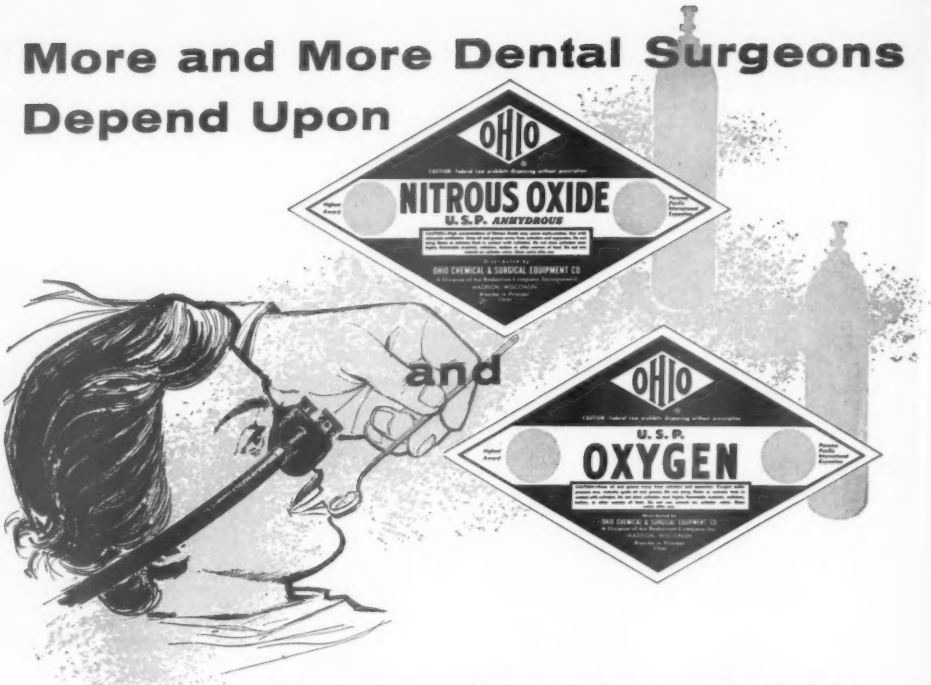

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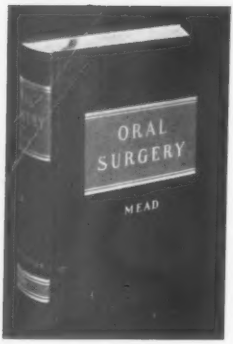

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Vol. 11, No. 12, December, 1958. Oral Surgery, Oral Medicine and Oral Pathology is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription rates: United States and its Possessions $15.00, Students $7.50; Canada, Latin-American, and Spain $15.50, Students $8.00; Other Countries $16.50, Students $9.00. Single copies $2.50 postpaid. Entered as second-class matter January 28, 1948, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in the U. S. A. Copyright © 1958 by The C. V. Mosby Company.

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THOMAS J. COOK

Practice Administration for THE DENTIST

Edited by **WILLIAM R. MANN, M.S., D.D.S.**, Ann Arbor, Michigan. Associate Professor of Dentistry, School of Dentistry, and Associate Director, W. K. Kellogg Foundation Institute; Graduate and Postgraduate Dentistry, University of Michigan.

and **KENNETH A. EASLICK, A.M., D.D.S.**, Ann Arbor, Michigan. Professor of Dentistry, School of Dentistry, University of Michigan.

1955, 386 pages, 6¾" x 9¾". PRICE, $8.50.

This volume provides a summary of the fundamentals necessary for the efficient, ethical practice of dentistry, for professional improvement, and for participation in community life—a long uncharted area of professional concern. It is the outgrowth of the workshop held at the University of Michigan. Those who attended represented 31 states, the District of Columbia, and four provinces of Canada. Instructors from 26 dental schools were in attendance.

At this workshop dentists, dental hygienists, dental assistants, dental teachers, and others interested in the subject critically presented the data, information, and opinions available regarding the entire area of practice administration and determined the best patterns for the dentist to adopt in organizing and administering his practice and for filling his niche of professional responsibility in his community.

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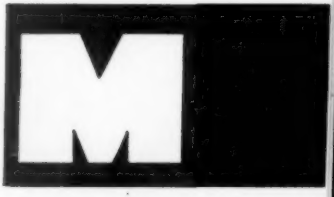

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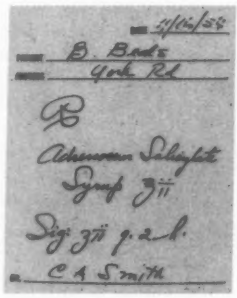

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The Management of ORAL DISEASE

A Treatise on the Recognition, Identification, and Treatment of Diseases of the Oral Regions

by

JOSEPH L. BERNIER, D.D.S., M.S., F.D.S., R.C.S. (Eng.) Colonel, Dental Corps, United States Army; Chief, Oral Pathology Branch, Armed Forces Institute of Pathology; Pathologist to the Registry of Oral Pathology of the American Dental Association; Professor of Oral Pathology, Georgetown University of Dentistry.

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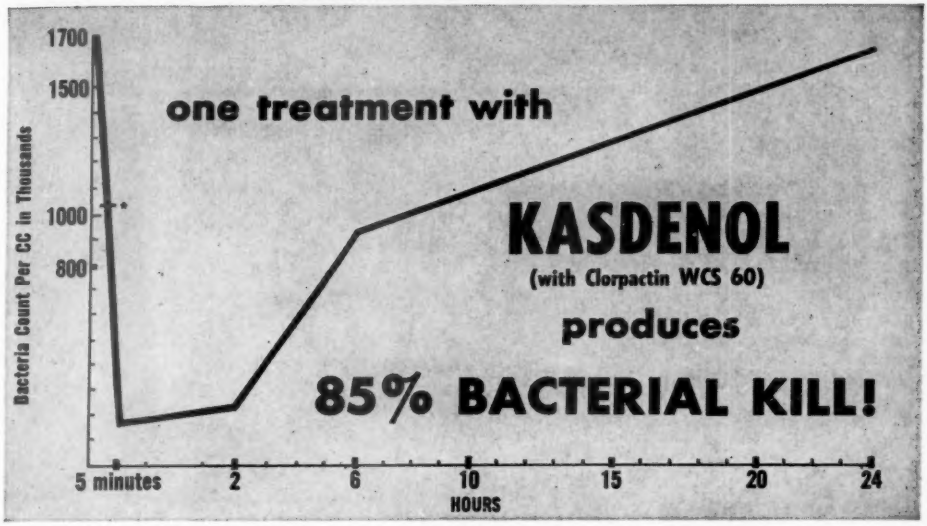

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